THE DIFFERENTIAL DIAGNOSIS OF MILD DEMENTIA AND DEPRESSION IN THE ELDERLY

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ORIGINALITY OF THESIS

I declare that this thesis reports my original work and that no part has been accepted or submitted for a degree or diploma at any University. To the best of my knowledge, no published or written material by another person has been included, except where due reference is given.

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ABSTRACT

Demented (n=8), depressed (n=8) and healthy elderly subjects (n=5) were given categorised and noncategorised words, over five trials, and tested under three retrieval processing conditions (free recall, cued recall, and recognition), in order to (1) examine the nature of memory deficits in dementia and depression, and (2) to test Weingartner's (Weingartner, Cohen, et al., 1981; Weingartner et al., 1981) cognitive model which claims that deficits experienced by demented and depressed patients arise from different types of memory failure.

Demented patients showed impairment for learning across trials for both categorised and noncategorised lists of words, compared to the depressed and control groups. However, the demented group retained more categorised than noncategorised words. Relative to healthy elderly subjects, demented patients were impaired on all retrieval conditions for both categorised and noncategorised word lists. Although significantly poorer than depressed subjects in freely recalling related words, dementia patients did not differ in freely recalling unrelated words, nor in recalling related words when given cues. For all three groups, recognition performance was superior to free recall and cued recall. Demented patients, however, were found to be significantly impaired on the recognition task, relative to the depressed group, for both categorised and noncategorised word lists.

Weingartner's hypothesis was not supported. For clinical purposes, recognition proved the task most likely to differentiate mild Alzheimer's disease patients from patients with depression. These effects were interpreted in terms of Hasher and Zack's (1979) theory of automatic and effortful processing, such that automatic tasks, such as recognition, which require less cognitive capacity, and which are therefore "easier", are impaired to a greater extent in demented compared to depressed subjects.

TABLE OF CONTENTS

INTRODUCTION

In 1985 there were 1,064,800 Australians aged over 70 years, of whom 290,000 were over 80 years. By the year 2000 these numbers will have increased to 1,623,400 people aged over 70, and 559,200 aged over 80, almost a doubling of the number of the very old (Grimes, 1988). Approximately eleven percent of the total Australian population will be aged 70 years or older by the end of the century. The vast numbers involved here draw attention to the role of the psychologist in accurately assessing mental health in the elderly.

Dementia and depression are among the most common disorders in the aged, and can exist as separate entities or may both be present in the same patient (Greenwald et al., 1989; Reifler, 1988; Reifler, Larson, & Hanley, 1982; Rovner, Broadhead, Spencer, Carson, & Folstein, 1989). The prevalence of dementia cases in Australia is increasing (Jorm & Korten, 1988). The number of dementia cases is expected to double every 5 years up to the age of 95 years (Jorm, Korten & Henderson, 1987). The prevalence of dementia ranges approximately from 5% in the 65 to 70 year age group, to 20% in those aged 80 years and over (Henderson, 1983). However, estimates of the prevalence of dementia in the general populations have varied according to the country studied (Henderson, 1983). This variation in the rate of prevalence across countries has been attributed to the variety of methodological procedures employed (Ineichen, 1987; Kay, 1988). The most common cause of dementia is Alzheimer's disease, which accounts for over half of all cases of dementia, and is a major cause of admission among geriatric patients to hospital (Grimes, 1988). The prevalence of depression is also high in the elderly. Recent Australian community surveys (Burvill, 1988) have shown that severe depressive illness occurs in 1.8% to 2.5% of the elderly population, with up to 13% having mild depression.

The differential diagnosis of dementia and depression has important practical consequences because of its implications for management, treatment and prognosis. For example, in terms of treatment, the symptoms of depression are generally reversible by the use of appropriate medication. While some dementias are treatable, most, such as Alzheimer's disease, are not. Although the differential diagnosis of dementia and depression is of considerable significance, clinicians often have difficulty distinguishing between depression and dementia, particularly in the early stages, since the disorders have overlapping symptoms (Marsden & Harrison, 1972; Nott & Fleminger, 1975; Ron, Toone, Garralda, & Lishman, 1979; Smith & Kiloh, 1981). For example, while memory impairment is an early and prominent aspect of cognitive decline in dementia (Hart, Kwentus, Taylor, & Harkins, 1987; McLean, 1987; Miller, 1975; Miller 1981; Sim & Sussman, 1962), it is also seen in depression (Hertel & Hardin, 1990; W.R. Miller, 1975; O'Connor, Pollitt, Roth, Brook, & Reiss, 1990). Moreover, depressed mood, anxiety, loss of interest, decreased spontaneity, somatic complaints and irritability are seen in early dementia (Liston, 1977; Liston, 1979; O'Connor et al., 1990; Sim & Sussman, 1962).

Diagnosis of dementia is also complicated by the fact that other mood disorders, apart from depression may mimic the syndrome of dementia. For example, pseudodementia may present symptomatically as dementia. The term pseudodementia has been described by Jorm (1986a) to refer to "cases where the features of dementia are closely mimicked, but the diagnosis has to be changed later because the subsequent course of the disorder involves a remission of the cognitive deficit (p.11)."

To further complicate the diagnosis, as already indicated, dementia and depression have been found to coexist, especially when the dementing disorder is at the mild to moderate stage (Greenwald et al., 1989; Lazarus, Newton, Cohler, Lesser, & Schweon, 1987; McAllister & Price, 1982; Reifler, 1986). Estimates of the prevalence of clinical depression among patients with dementia have varied from 11% (Greenwald et al., 1989) to 57% (Liston, 1979). It is likely that this wide variation results from the application of a variety of diagnostic methods and criteria.

In the attempt to achieve differential diagnosis it has been suggested the presence of aphasia (disorder of language), apraxia (inability to carry out voluntary motor functions), and agnosia (failure to recognise or identify objects) may indicate dementia. However, these features are not obvious until the middle stage of the dementing disorder (Corsellis, 1976). Moreover, aphasia, apraxia and agnosia are not universal in Alzheimer's disease, even in the later stage (e.g., Breitner & Folstein, 1984; Knesevich, Roro, Morris, & LaBarge, 1985). Thus, these basic distinguishing features of dementia and depression are of little value as diagnostic aids in the crucial early stages of dementia. The major problem remains that of differentiating those patients who are suffering from depression (or dementia and mood disorder) from those patients with a dementing process.

The clinical history may help to differentiate early dementia and depression on the basis of historical information including duration, mode of onset, and character of the early symptoms. The variable and uneven nature of the cognitive impairment, rapid progression of symptoms, and a past history of depression (Huppert & Tym, 1986), may lend support to a diagnosis of depression. Abnormalities of mood in dementia are less frequent and, when present, less pervasive than in depression. Table 1 is an example of an attempt to summarise the distinguishing clinical features for dementia and depression as compared to normal ageing, and is adapted from Burvill (1988).

Table 1

A Summary of
Clinical Features for Dementia, Depression and Normal Ageing

	Dementia	Depression	Normal Ageing
1. Current Symptoms: a. Complaints	Reported by others; patient often unaware.	Patient usually complains of memory problems.	Patient may complain of memory loss.
b. Types of memory problems reported	Major—interfere with activities of daily living.	Mild, mostly due to inattention.	Mild increase in normal forgetting.
c. Hallucinations and delusions	Paranoid accusations sometimes present.	Absent, except in severe cases.	Absent.
2. History a. Onset	SDAT—insidious Multi-infarct—sometimes sudden.	Coincides with life changes. Onset often abrupt.	Reactions to normal life changes.
b. Duration	Months or years.	At least two years.	
c. Progression	SDAT—gradual. Multi-infarct—stepwise.	Not progressive.	Minimal over long periods of time.
d. Fluctuation	SDAT—little. Multi-infarct—some daily fluctuation. Usually worse in evening.	Typically worse in the morning.	Mild situational fluctuations.
e. Anxiety	Often.	Prominent.	
f. Agitation or Retardation	Variable.	Severe cases.	
g. Conscious State	Clear.	Clear.	Clear.

(adapted from Burvill, 1988).

Although useful as a guide, Table 1 oversimplifies the diagnostic issues and, alone, is not a satisfactory basis on which to make a diagnosis. That the problem of differential diagnosis is not a trivial one, is evident from the high frequency of misdiagnoses which has been found upon follow-up. For example, Marsden and Harrison (1972) reviewed patients admitted to hospital thought to be suffering from primary dementing illness. On reassessment, 8% were found to be cases of depression. In a similar Australian study, Smith and Kiloh (1981) found that 5 % of patients admitted to hospital for dementia, were misdiagnosed depressives. Nott and Fleminger (1975) enquired into the long-term fate of a group of 50 patients diagnosed as suffering from presenile dementia (i.e., onset prior to age 65 years). Of the 35 patients they were able to trace, less than half were found to be demented at follow-up. Most of these misdiagnosed patients were found to have personality disorders and neurotic illnesses. Ron et al. (1979) followed up cases diagnosed as having pre-senile dementia. Five to fifteen years later nearly a third (31%) were judged to have been misdiagnosed.

Clinical diagnosis would be made easier if there were some qualitative differences in the type of cognitive deficits exhibited by demented and depressed patients. For example, if it were found that the type of memory deficit observed in depression differed in kind from that of dementia, differential diagnosis would be facilitated, even in the early stages.

The aim of the present thesis is two-fold: (1) to examine the nature of memory deficits in depression and dementia; and, (2) to test a cognitive model of memory in which dementia and depression are seen as arising from different types of memory failure. In the following sections of this chapter, the nature of dementia and depression and their associated cognitive deficits are reviewed. Following this, theoretical accounts of the types of memory failure in dementia and depression are considered. However, before examining memory deficits in the clinical groups, it is important to review briefly the type of deficit observed in healthy elderly subjects, so that the pathological memory changes may be presented within the context of normal age related changes.

1.2 EFFECTS OF AGEING ON INTELLIGENCE AND MEMORY

Research into age related decline in memory has focussed upon the distinctions among episodic, semantic and procedural memory (Mitchell, 1989). There is evidence in old age of impairment in episodic memory, but not semantic or procedural memory (Butters, Granholm, Salmon, Grant, & Wolfe, 1987; Cermak, 1984; Jacoby, Baker, & Brooks, 1989; Mitchell, 1989; Mitchell & Perlmutter, 1986). These terms will be defined here in some detail because distinctions in these types of memory are used later to describe the deficits observed in dementia and depression.

1.2.1 Definitions of Episodic, Semantic and Procedural Memory

Episodic memory involves conscious recollection for "personally experienced events and their temporal relations" (Tulving, 1985, p.387). The primary measures employed to study episodic memory include recognition, free recall and cued recall. Semantic memory is also available to consciousness, but, unlike episodic memory, is not tied to spatial and temporal autobiographical contexts. Tulving described semantic memory as a "mental thesaurus" (Tulving, 1972, p.386). More precisely, "semantic memory is an organised store of knowledge that a person possesses about words, concepts and their associations, and the rules for manipulating those symbols and concepts" (Tulving, 1985, p.388). Semantic memory is commonly assessed through lexical decision and naming tasks (semantic priming). In naming tasks, the time taken to name a word preceded by a semantically related word is measured. Words preceded by semantically related words are named faster than words preceded by nonsemantically related words.

Procedural memory has also been described as "implicit memory" (Graf & Schacter, 1985), and "memory without awareness" (Jacoby & Witherspoon, 1982). Procedural memory allows a person to make learned, overt responses in the context of particular stimuli and is therefore, "prescriptive rather than descriptive: It provides a blueprint for future action without containing information about the past" (Tulving, 1985, pp.387-388). Schacter (1985) defines memory performance on an "implicit" test as that "which does not demand conscious recollection of a learning episode" (p.41). Procedural memory tasks often involve a second presentation of a previously experienced stimulus. Primary measures of implicit memory include repetition priming (Graf, Shimamura, & Squire, 1985; Shimamura, 1986), or priming for new associations (Shimamura & Squire, 1984). In addition, word fragment completion tasks (Schacter & Graf, 1986a, 1986b; Tulving, Schacter, & Stack, 1982) and word stem completion (Greene, 1986; Salmon, Shimamura, Butters, & Smith, 1988; Shimamura, Salmon, Squire, & Butters, 1987), which require the subject to complete either three-letter word fragments, or the missing part of a hyphenated word, from items of a previously studied list, are used to measure procedural or implicit memory. Subjects are instructed to complete the item with the first word that comes to mind. Another example of a measure of procedural memory is homophone spelling (Jacoby & Witherspoon, 1982; Schacter, 1985), where the subject is asked a number of questions which contain homophones which are spelt differently, depending on the meaning of the sentence. A homophone is a pair of words which are pronounced the same but are spelt differently (e.g., reed, read). For example, a subject may be asked to name a musical instrument that employs a reed. The subject is then required to spell the homophone in a subsequent spelling test.

1.2.2 Age-Associated Changes in Memory

Mitchell (1989) has summarised the major findings with respect to changes in episodic, semantic and procedural memory in old age. Episodic memory shows the greatest changes in old age (e.g., Craik, 1977; Mitchell & Purlmutter, 1986; Purlmutter & Mitchell, 1982; Smith, 1980). Various studies have investigated retrieval from semantic memory in the aged (Bowles, Obler, & Albert, 1987; Drachman & Leavitt, 1974; Mitchell, 1989; Weingartner, Grafman, Boutelle, Kaye, & Martin, 1983). Evidence from these studies suggests that retrieval from semantic memory either remains stable with increased age, or even improves. Finally, a summary of findings from ageing studies suggests that procedural memory may be immune to age-related decline in cognitive functioning (Graf & Schacter, 1985; Jacoby & Witherspoon, 1982; Mitchell, 1989; Moscovitch, Winocur, & McLachlan, 1986).

Thus, in summary, while normal elderly subjects seem to show impairments on certain types of memory tasks, many aspects of memory do not show decline. It is also important to note that many of the episodic deficits shown by elderly people are reversible or modifiable. For instance, recognition and cued recall performance in older subjects (typically aged 60-80 years) often approaches the levels of performance of young subjects (Craik, 1977). Recognition and cued recall tasks are thought to remain relatively age immune because the level of cognitive operations required is substantially reduced, owing to the supportive nature of environmental cues or guidelines (Craik, 1984). As the task is less supported at retrieval or encoding, larger age differences become manifest, as for instance, in unstructured free recall tasks. Craik and Rabinowitz (1984) have suggested that these findings are consistent with the idea that older people may have fewer processing resources available to carry out mental operations.

Craik (1984) considers that a reduction in processing resources would interfere with the person's ability to achieve effective, efficient encoding and retrieval operations. Craik further suggests that older subjects, given their reduced processing capacity, fail to actively modify novel situations because of the difficulty and effort involved. Active manipulation of a task is considered to require sustained mental thought, or "effortful" processing (Hasher & Zacks, 1979), which becomes increasingly difficult to activate, with fewer processing resources available. Thus, according to Craik (1984), decline in memory tasks of the elderly is related more to the type of task involved, whether that task requires highly practised skills, or unfamiliar problem-solving skills, than to a separate body or system of memory, such as episodic memory.

The nature of dementia and depression will now be reviewed. Following this, the memory deficits associated with dementia and depression will be described in detail.

1.3 NATURE OF DEMENTIA

Dementia is a syndrome or group of symptoms characterised by an acquired persistent and usually irreversible impairment of intellectual ability, memory and language, with at least one of the following changes: impaired judgement, impaired visuospatial skills, or changes in personality or emotional state (DSM-111-R, 1987). Historically, the term "dementia" has been used in a variety of ways, and, although there is consensus about the major features of dementia, definitions vary somewhat.

Lishman (1978) defines dementia as "an acquired global impairment of intellect, memory and personality, but without loss of consciousness." The stipulation that the intellectual impairment must be acquired distinguishes dementia from the congenital mental retardation syndromes (Cummings & Benson, 1983). A more detailed definition is given by the Royal College of Physicians (1981):

"Dementia is the global impairment of higher cortical functions, including memory, the capacity to solve the problems of day-to-day living, the performance of learned perceptuo-motor skills, the correct use of social skills and control of emotional reactions, in the absence of gross clouding of consciousness (p.4)."

A third definition and a set of criteria for diagnosing dementia is provided by the American Psychiatric Association Diagnostic and Statistical Manual of Mental Disorders-Revised (DSM-III-R) (1987). These criteria are widely used in clinical psychological practice. The DSM-III-R lists the following criteria which must be present for dementia to be diagnosed:

(a) "Demonstrable evidence of impairment in short-and-long-term memory;

(b) A loss of intellectual abilities which is severe enough to interfere with social and occupational functioning;

(c) At least one of the following:

  (1) Impairment in abstract thinking, as indicated by inability to find similarities and differences between related words, difficulty in defining words and concepts, and other similar tasks;

  (2) Impaired judgement;

(3) Other disturbances of higher cortical function, such as aphasia (disorder of language), apraxia (inability to carry out motor activities despite intact comprehension and motor function), agnosia (failure to recognise or identify objects despite intact sensory function), 'constructional difficulty' (e.g., inability to copy three-dimensional figures, assemble blocks, or arrange sticks in specific designs);

(4) Personality change, that is, alteration or accentuation of premorbid traits;

(d) Not occurring exclusively during the course of delirium;

(e) Either (1) or (2):

(1) Evidence from the history, physical examination, or laboratory tests, of a specific organic factor that is judged to be aetiologically related to the disturbance;

(2) In the absence of such evidence, an aetiologic organic factor can be presumed if the disturbance cannot be accounted for by any nonorganic mental disorder (e.g., Major Depression accounting for cognitive impairment) (p.107)."

The DSM-III-R (1987) also provides guidelines for diagnosing the severity of dementia:

In "mild" dementia, work or social activities are significantly impaired, although the capacity for independent living remains, as does adequate personal hygiene and relatively intact judgement. In moderate dementia, independent living is hazardous, and some degree of supervision is necessary. In "severe" dementia, activities of daily living are so impaired that continual supervision is required (e.g., unable to maintain minimal personal hygiene; incoherent or mute).

1.3.1 Common Causes of Dementia and Associated Pathology

Numerous disorders can produce dementia in the elderly. However, three disorders, Alzheimer's disease, Multi-infarct dementia (MID) and Alzheimer's disease and MID combined, account for most cases. According to Jorm and his colleagues' (1987) review of prevalence studies of Alzheimer's disease and Multi-infarct dementia, Alzheimer's disease is the most common cause of dementia in Western European countries. Until 1970, the term Alzheimer's disease was used to refer only to dementia affecting people under the age of 65. However, a study by Tomlinson, Blessed, and Roth (1970) established that senile patients showed the same brain changes as the younger patients who had been diagnosed as suffering from Alzheimer's disease. Since then, the term "senile dementia of the Alzheimer type" (SDAT) has been used to describe both presenile and senile forms (Henderson & Jorm, 1986). The course of dementia is not, as yet, well described, although the neuropathological changes that accompany it are well known.

Alzheimer's disease is characterised by a widespread functional disturbance of the human brain. Grossly, the brains of severe dementia cases at death are atrophic, often weighing less than 1000 grams (Corsellis, 1970). The atrophy is most pronounced in the parietal, temporal, and frontal areas with the occipital and motor regions being largely spared (Filley, Kelly, & Heaton, 1986). The most distinguishing features of Alzheimer's disease are the microscopic changes in the distribution of senile plaques and neurofibrillary tangles, the pattern of cell loss, and the neurotransmitter deficits. Each is described below.

Senile plaques consist of a core of the abnormal starch-like protein, amyloid (Kang et al., 1987). The gene carrying this protein is localised on chromosome 21.

Kang and his colleagues have suggested that a double amount of the amyloid protein, accounts for the increased numbers of plaques and tangles found in the brains of demented people. The other classic neuropathological changes are neurofibrillary tangles, which are composed of bundles of paired filaments wound around each other in a helical pattern. They occur within nerve cells and gradually take over much of the cell space (Riekkinen, Laulumaa, Sirvio, Soininen, & Helkala, 1987; Roth, 1986). The number of plaques and tangles observed at autopsy is known to be strongly correlated with the severity of the dementia before death (Blessed, Tomlinson, & Roth, 1968). Similar neuropathological changes have been discovered in the brains of patients with Down's syndrome. Heston (1984) and Wisniewski, Wisniewski, and When (1985) studied the brains of Down's syndrome patients and concluded that all, or nearly all, Down's cases over the age of 40 years developed the neuropathological changes characteristic of Alzheimer's disease. Because Down's individuals carry an extra copy of chromosome 21, they will also have an extra version of the amyloid gene which lies on this chromosome. It is thought that this factor is responsible for the same changes found in Alzheimer's disease patients.

In Alzheimer's disease these histological changes are found throughout the cerebral cortex, and in much greater numbers in the hippocampus (Rossor 1982; Roth et al., 1986). Ball and his colleagues (1985) have shown that the posterior half of the hippocampus is particularly affected. The finding of the hippocampal involvement provides an explanation of the great difficulty Alzheimer patients have in learning new material, since damage in a variety of hippocampal areas has been found to yield disorders which O'Keefe and Nadel (1978) term as "limited amnesias". The authors include within this terminology the disorders of agnosia, apraxia, certain forms of aphasia, proposagnosia, which is a specific deficit in face memory, and selective deficits in verbal short-term memory.

Since the discovery of a cholinergic deficit in Alzheimer's disease in 1976 and 1977, there has been a great deal of interest in the neurotransmitter changes in dementia, although the initial hope for a treatable deficit (analogous to the dopamine deficit in Parkinson's disease), has not been realised. Three independent laboratories have reported lowered activity of the enzyme choline acetyltransferase (ChAT) in post-mortem cerebral cortex from Alzheimer cases (Bowen, Smith, White, & Davison, 1976; Davies & Maloney, 1976; Perry, Perry, Blessed, & Tomlinson, 1977). Neurons which use the cholinergic system have been shown to use the enzyme ChAT to manufacture the neurotransmitter acetylcholine. In contrast to acetylcholine, ChAT has been found to be relatively stable after death, providing a valuable post-mortem marker of cholinergic neurons (Mann & Yates, 1986; Rossor, 1982). The most marked reductions in ChAT activity have been seen in the temporal neocortex, hippocampus, and amygdala (Tyrrell & Rossor, 1988). The ChAT activity has been found to be correlated with the density of senile plaques and neurofibrillary tangles and with the severity of dementia, with those patients dying at a younger age showing greater severity of cholinergic abnormality. No cholinergic deficit has been found in the frontal lobes of patients dying after the age of 80 years. Furthermore, within the basal forebrain the reduction in ChAT activity is confined to the area of the nucleus basalis of Meynert, believed to be the major source of cortical cholinergic innervation. Neurons in this region send long ascending fibres to the hippocampus and cortical regions. Reduced cell counts within the nucleus basalis of Meynert from deceased Alzheimer patients have been found, indicating that cell loss may be due to degeneration, subsequently leading to reduced levels of the enzyme ChAT, reducing the neurotransmitter acetylcholine (Jorm, 1987; Mann & Yates, 1986; Whitehouse et al., 1982). Cholinergic reduction has not been found in the adjacent putamen and globus pallidus, providing evidence that the cholinergic abnormality is not generalised but predominantly affects the ascending projection system.

The significance of the cholinergic abnormality in Alzheimer's disease, and its role in memory consolidation (Weingartner, Sitaram, & Gillin, 1979), has gained firmer ground with studies that show a similar impairment of memory using normal subjects and anticholinergic drugs. One such drug, scopolamine, a cholinergic receptor blocker, has been the focus of intense investigation. Scopolamine disrupts acetylcholine by blocking presynaptic receptors so that they remain insensitive to acetylcholine. Administration of scopolamine produces a transient amnesic disorder in normal subjects, similar in some, but not all, aspects to that demonstrated by demented patients. The Caine, Weingartner, Ludlow, Cudahy, and Wehry (1981) study found that subjects receiving the drug, scopolamine, showed impairment of acquisition and retrieval despite normal immediate memory span. These investigators (Caine et al., 1981; Drachman & Leavitt, 1974; Weingartner et al., 1979) hypothesise that scopolamine influences the transfer of information from short-term to long-term memory storage.

The serotonin, noradrenaline, and dopamine neurotransmitter systems which extend to the cerebral cortex, have also been reported to be abnormal in Alzheimer's disease (Rossor, 1982). The examination of neurotransmitter-specific populations of cerebral cortical neurons, such as somatostatin, have been found at greatly reduced levels in the hippocampus, and frontal cortex. In addition, neurons using this neurotransmitter appear to be affected by plaques and tangles (Beal et al., 1985). These findings may reflect degeneration of postsynaptic neurons or cortical afferents in the patients' cerebral cortices, interfering with the processes of memory. It is thus likely that many neurotransmitter systems contribute to the memory deficits observed in Alzheimer's disease.

Multi-infarct dementia (MID), so named by Hachinski, Lassen, and Marshall (1974) because its cause is due to multiple strokes or infarctions, is another common cause of dementia. This type of dementia is also referred to as vascular dementia, because of its association with the vascular system. Multi-infarct dementia is the second most common cause of dementia in Western European countries, and the most prevalent in Japan and Russia (Jorm et al., 1987). A stroke results from multiple vascular occlusions, or blockages within the arteries, which prevent the flow of blood supply to specific areas of the brain, causing death to the surrounding nerve cells (Heston & White, 1983). Sometimes these small strokes are caused by pieces of plaque on the arterial wall breaking away and travelling to the brain, where they cause a blockage (Hachinski et al., 1974). Like Alzheimer's, multi-infarct dementia is progressive. However, in contrast to Alzheimer's disease, vascular dementia may progress in a series of small steps over time, followed by a plateau of mild remission, which is followed by further deterioration (Mahendra, 1984). Vascular dementia affects cortical and subcortical regions of the brain. Cortical infarcts are said to result in aphasia, amnesia, and visuospatial disturbances, and multiple subcortical infarcts in Psychomotor retardation, memory disorders, and cognitive impairment (Cummings & Benson, 1988). Personality is relatively well preserved, although severe depression is common to this type of dementia (Hachinski et al., 1974), possibly due to the preservation of a considerable degree of awareness, or insight, into the person's own condition (Mahendra, 1984). In 1975, Hachinski et al. developed the Ischaemic Score as one method of differentiating multi-infarct dementia from other disorders, such as Alzheimer's disease. Characteristic features include a history of hypertension, previous strokes, diabetes, and, in contrast to Alzheimer's disease, an abrupt onset of cognitive deficit (Cummings & Benson, 1983).

The third common cause of dementia is a combination of multi-infarct dementia and Alzheimer's disease. Because both disorders become increasingly frequent with age, it is not surprising that they may co-occur in the elderly. This combined disorder is referred to as "mixed dementia".

A distinction on the basis of neuroanatomical areas believed to be damaged in dementia has also been made in an attempt to understand the basis of behavioural and psychological deficits demonstrated by dementia patients. For example, Alzheimer's disease and Pick's disease are believed to be associated with impairment in the cortical structures and are classed as cortical rather than subcortical dementias. Multi-infarct dementia may affect both cortical and subcortical structures (Cummings and Benson, 1988). Other less common causes of dementia are Huntington's and Parkinson's disease, which are classed as subcortical dementias. Because dementia consists of a number of heterogeneous conditions, it is important that patients are carefully selected before inclusion in empirical investigations. Each sub-type of dementia may be characterised by a different type of cognitive impairment and combining patients for research purposes may not be appropriate.

1.4 MEMORY DEFICIT IN DEMENTIA

Dementia is associated with a range of deficits in many areas of mental functioning. Memory difficulties are the most prominent. In the following section semantic deficits will be described first, followed by a description of procedural and episodic memory deficits in dementia.

1.4.1 Semantic Memory Functioning in Dementia

Semantic memory deficits have been observed in dementia using naming, sentence construction, word fluency and priming tasks. A number of authors have described the difficulties that dementia patients have in providing the names of common objects or concepts. Barker and Lawson (1968) found that senile patients were impaired in naming objects with low-frequency names as compared to objects with high-frequency names. Bayles (1982) found that demented subjects, whilst showing difficulty in correctly naming an object, more frequently tended to name or describe something associated with the stimulus item, such as "sweeping-up" for the test item "vacuum cleaner". Schwartz, Marin, and Saffran (1979) reported a subject who could demonstrate the use of objects, but had lost the ability to name them. Allison (1962) and Gustafson, Hasberg, and Ingvar (1978) showed that object-naming tasks presented less difficulty than more abstract word-finding tasks, such as, finding similarities or opposites, sentence completion, or word fluency. Paraphasias (incorrect and inappropriate words in a sentence) are reported to be frequent, with either phonetic or semantic substitutions. Thus, for example, a patient may use the word "firebugs" for matches. Other language difficulties have been reported. Perseveration is common and may occur in various forms, such as simple repetition of the same word or syllable (Bayles, Tomoeda, and Kasznaik, 1985). Intrusions (nonlist items) are also reported with varying frequency (Appel, Kertesz, & Fisman, 1982; Bowles et al., 1987; Fuld, Katzman, Davies, & Terry, 1982).

Despite the failure in word naming, or in generating word names, one aspect of language has been found to be relatively well preserved. Syntactic knowledge, that is, the ability to properly connect words in a sentence, can remain intact in senile dementia, even in the later stages of the disease (Bayles, 1982; Caramazza & Berndt, 1978; Kempler, Curtiss, & Jackson, 1987; Martin & Fedio, 1983; Miller, 1981). The preservation of syntactical aspects of language contrasts with the loss of semantic aspects of language. For example, demented subjects can correct syntactically abherrant sentences ("she lost John book"), but cannot correct semantically abherrant sentences ("she lost John's temper")(Huppert & Tym, 1986; Schwartz et al., 1979). Jorm (1986b) accounts for this phenomenon by proposing that the uneven developmental course of progressive cognitive decline can be understood in terms of the controlled and automatic information processing model as developed by Shiffrin and Schneider (1977). Jorm suggests that syntactic knowledge is more overlearned and automatic, while semantic knowledge requires attentional resources for its application because of its less predictable nature. Controlled processing, which requires the attentional resources of the individual, is seen to decline early in the disorder, while automatic processing, which does not require additional resources, remains unaffected until the late stages of the disease.

Major deficits are seen in searching semantic categories, and can be demonstrated on verbal-fluency tests which require the retrieval of words from a particular semantic category, such as names of animals (Bayles, 1982; Bayles & Tomoeda, 1983; Butters et al., 1987; Martin & Fedio, 1983; Ober, Dronkers, Koss, Delis, & Friedland, 1986; Weingartner et al., 1981). Semantic memory deficits are, however, less evident where subjects are not required to generate examples or actively search semantic memory.

Results from priming studies (Nebes & Brady, 1988; Nebes, Boller, & Holland, 1986; Nebes, Brady, & Huff, 1989; Nebes, Martin, & Horn, 1984) suggest that at least some components of semantic structure and function are spared by Alzheimer's disease. In contrast to findings by Martin and Fedio (1983), Nebes and

Brady (1988) found that Alzheimer patients were capable of recognising the relationship between a concept and its various attributes. The subject was presented with a test object, followed by ten stimulus words, five of which were related to the test item. Relationship to the test item was based upon its category, function, a feature, and an associate of the test object. For example, for the test objects "shirt", the function would be to "wear", the feature, "collar", and the associate, "tie". The subject was asked to respond to these stimulus words if they made him think of the test object. In comparison to healthy old and young subjects, demented patients were found to be no slower in determining whether a target concept was related to a specific attribute.

Dementia patients appear to be able to use semantic information in less effortful, or more automatised tasks. For instance, when asked to complete a letter fluency task, such as giving as many different words as possible beginning with the letter "F", Butters et al. (1987) found that mild Alzheimer patients generated nearly as many correct responses as the elderly control subjects, in comparison to category fluency tasks (e.g., generating as many words as possible in the category of animals). Butters and colleagues explain this sensitivity to the category task, by suggesting that the letter fluency task can be performed using phonemic cues to search an extensive knowledge base, whilst the category task demanded a search for examplars of a specific category, thus requiring greater cognitive "effort" to successfully complete.

In addition, semantic context, both in the form of single words and of entire sentences, was found to influence word-naming to the same degree in normal and demented subjects (Nebes et al., 1984). The semantic priming task in this study, was used to see whether the associational links between semantic concepts remained intact in Alzheimer's disease patients. In a semantic priming task, the measurement used is the time taken for the subject to respond to previously presented semantically related words, as for example, membership in a common category, such as the paired words, "doctor-nurse". This response time is then compared to the time taken to respond to unrelated paired words, as for example, the paired words, "pepper-goat". If the time taken to respond correctly to the related stimuli decreases by comparison to unrelated stimuli, then this is assumed to reflect more efficient processing of the material by the aid of priming with associated pairs of words. That is, the subject is responding at a faster rate by linking one stimulus with another of the same category, and therefore it must be assumed is influenced by an intact knowledge of semantic relationships.

A second study by Nebes et al. (1989) compared automatic and attention-dependent priming in dementia patients, by comparing the effect of single word primes on a lexical-decision and a word-naming task. In the lexical-decision task, subjects were required to determine whether a given string of letters comprised a word in the English language. In the word-naming task the subjects were required to name the words that were presented visually. This latter task required a less attention-dependent process for successful completion. No significant difference was found in the performance for either the attention-dependent or the more automatic task. Thus, the hypothesis that semantic priming in demented patients was due solely to automatic processes was not proved. Nebes et al. (1989) suggests that one possible explanation for the pattern of semantic deficits seen in dementia patients, may be the differentiation in intentional and incidental retrieval. Intentional retrieval requires the subject to actively search for information, using self-generated retrieval cues. By comparison, incidental retrieval occurs when the subject uses the stimulus material and task situation to guide memory access. Nebes (1989) postulated that intentional processes may be impaired in dementia.

1.4.2 Procedural Memory Functioning in Dementia

Despite impairments in many aspects of semantic memory function, procedural memory functioning appears to be preserved relatively well in dementia. Both skilled learning, simple classical conditioning, and repetition priming are types of procedural memory that appear to remain intact, at least in the early stages of the disorder. Learning in procedural memory has been called "implicit learning", or "learning without awareness" (Jacoby & Witherspoon, 1982), and refers to motor, perceptual and even cognitive skills that are acquired in progressive stages of consistent practice of the skill itself, and are not dependent on prior personal experience of the skill (Cohen & Squire, 1980; Eslinger & Damasio, 1986). As noted above, procedural memory can be assessed by priming for new associations (Moscovitch et al, 1986), and repetition priming (Shimamura, 1986).

A number of studies have provided evidence that dementia patients not only acquire, albeit unconsciously, information, but can learn completely new skills. Eslinger and Damasio (1986) found that Alzheimer patients failed to remember a list of common words and unfamiliar faces, but were able to demonstrate a learning curve similar to that of control subjects when they learnt a rotary pursuit motor skill. Corkin et al. (1986) found a dissociation in Alzheimer patients of verbal priming and skill learning. On tests of motor skills learning (bimanual tapping and rotary pursuit), the demented group demonstrated a learning curve, with substantially diminished scores for verbal priming tasks. Knopman and Nissen (1987) used a visual reaction time paradigm to test stimulus-response learning in Alzheimer patients. The demented group learned the procedure required for increasingly efficient responses without awareness of, or ability to explicitly explain, the sequence.

Evidence that procedural memory is relatively intact in dementia patients is supported by other researchers. Morris, Wheatley, & Britton, (1983) using yes-no recognition, free recall and word stem completion (the first three letters of each word), found word stem completion relatively unimpaired in dementia patients. Miller (1975) found no significant difference in word-stem completion performance of Alzheimer patients, compared to control subjects, but found they were significantly impaired on tests of recognition memory and free recall. In addition, Partridge, Knight, and Feehan (1990), using essentially the same word completion task as Salmon et al. (1988) and Shimamura et al. (1987), demonstrated that senile dementia patients showed normal word completion performance, relative to control subjects, whilst the free and cued recall tasks were impaired.

1.4.3 Episodic Memory Deficits in Dementia

Episodic memory is severely impaired in dementia and episodic memory deficits are the earliest symptoms of dementia. The initial memory disturbance in Alzheimer's disease is characterised by impaired ability to learn new material, both verbal and visuospatial. In clinical practice, episodic memory is often divided into primary memory, and secondary memory. Primary memory (sometimes referred to as working or short-term memory)(Baddeley, 1986; Moscovitch, 1984), refers to memory for events or material lasting for as long as approximately 30 seconds (Morris & Baddeley, 1988). It serves as a limited capacity store from which information is transferred to a more permanent store. Primary memory relies heavily on continuous attention on the material to be encoded (Morris & Baddeley, 1988). While some authors use the terms "primary memory" and "working memory" interchangeably, primary memory has been distinguished from working memory by Craik and Rabinowitz (1984). For these authors, primary memory pertains to a situation in which small amounts of material are held briefly in memory, but are not transformed in any way.

Examples include digit span, and the recency effect in a free recall task. By contrast, working memory is assumed to involve the subject in actively holding, manipulating, and transforming the material in memory over a brief period before making a response. Examples include backward digit span, and various dual-task paradigms. Secondary memory, also referred to as long-term memory (Moscovitch, 1984), refers to the person's ability to store information in a more permanent store. The process of storing information into secondary memory is called consolidation.

The testing of episodic memory covers three distinct activities, namely, acquisition, storage, and the ability to retrieve information learned in the past. Evidence concerning each of these phases will be considered in turn.

Acquisition

Alzheimer patients typically show deficits in performance of primary memory tests, such as the recency component of free recall, memory span, and the Brown-Peterson test, a measure of short term forgetting following distraction (Morris & Kopelman, 1986). Alzheimer patients typically remember only the most recently presented items in a free recall task (Martin & Fedio, 1983; Wilson, Bacon, Fox, & Kaszniak, 1983). Memory span may also be reduced (Miller, 1971), although patients in the very early stages of dementia may show no decrements (Weingartner et al., 1981). Normal elderly can retain from six to seven digits in primary memory, whilst mild to moderately impaired Alzheimer patients usually only manage approximately five digits (Kopelman, 1985).

Researchers have also demonstrated deficits in the Central Executive functioning of working memory (Baddeley & Hitch, 1974), in which the capacity to perform simultaneously two concurrent tasks is impaired in the demented patient

(Baddeley, Logie, Bressi, Della Sala, & Spinner, 1986; Becker, 1988; Morris & Baddeley, 1988). Morris (1986) used a variety of distractor tasks, based on the assumption that the more demanding the distractor task, the greater proportion of available processing resources were used. Morris ranged his distractors from none to difficult. With no distraction, Alzheimer patients were able to remember the material for as long as 20 seconds. As the distractor tasks increased in difficulty, the amount of information acquired, was reduced. Corkin (1982) demonstrated that mild, moderate and severe Alzheimer patients showed impaired primary memory, using the Brown-Peterson distractor task. Dementia patients also failed to show a recency effect in the serial position curve of a free recall task (Miller, 1971), providing further evidence of poor primary memory.

Secondary memory is also impaired in Alzheimer's disease, but perhaps with greater severity than primary memory (Morris & Kopelman, 1986). Deficits are found in learning new verbal and nonverbal material using recall, recognition and cued recall retrieval procedures (Corkin, 1982; Kopelman, 1986; Miller, 1971). Butters et al. (1987) showed that when asked to recall short story passages, Alzheimer patients remembered few correct facts, and made numerous intrusions (nonstory items). Miller (1971) found an almost complete absence of a primacy effect in the serial position curve of a free recall task in Alzheimer patients. Words recalled from the beginning of a list (primacy effect) are assumed to reflect those words which have been successfully transferred through the long-term memory store. Thus, an absence of a primacy effect is indicative of an impairment in long-term memory in dementia. Corkin (1982) demonstrated Alzheimer patients to be impaired on both verbal and nonverbal paired associate learning. These deficits were particularly marked with words of low associability (e.g., bottle-comb). Deficits for learning material have been noted for learning new information (La Rue, 1989), when using the selective reminding procedure for verbal learning (Buschke, 1973; Hart, Kwentus, Hamer, & Taylor, 1987), and for learning semantically organised and nonorganised word lists (Weingartner et al., 1981), in which study Weingartner showed that dementia patients failed to benefit from list clustering compared to normals.

Storage

Although the evidence is clear that dementia patients have great difficulty in acquiring information, it has been claimed that once initial learning has been accomplished, Alzheimer patients showed a normal rate of forgetting. For instance, work by Becker, Boller, Saxton, and McGonigle-Gibson (1987) found that Alzheimer patients did not forget at a faster rate than normal elderly subjects over a 30 minute retention interval, although they did recall substantially less information. In addition, Huppert and Kopelman (1989) studied the rate of forgetting for demented patients of visuospatial material, and found that, although acquisition was much slower, rate of forgetting was similar to that of normal elderly subjects. The issue is yet to be resolved, however, since conflicting findings have been reported by Hart, Kwentus, Taylor, et al. (1987). These researchers showed rapid forgetting in the first 10 minutes after learning to criterion, in comparison to depressed subjects.

Retrieval

There is some evidence that cues at the time of retrieval may aid memory performance in dementia patients. However, the extent to which cues at retrieval are effective varies according to the type of cue used. That is, the evidence suggests that "structural cues" may be effective in aiding memory, but that "semantic cues" may not be as effective.

Davis and Mumford (1984) in the only direct comparison study, demonstrated that cueing the patient with the word's first letter was more effective than cueing with the name of its semantic category. In a study examining the effectiveness of "structural cues", Morris et al. (1983) demonstrated that cued recall procedure using the first three letters of a word, successfully improved memory performance. Although there has been one contradictory finding (Buschke, 1984), a number of investigators have demonstrated that "semantic cues" are relatively ineffective in improving memory performance in dementia patients, compared to normal elderly subjects (e.g., Cushman, Como, Booth, & Caine, 1988; Tuokko & Crockett, 1989; Weingartner et al. 1981). It is significant that the Buschke (1984) study contained only four subjects.

Recognition performance is also impaired in dementia patients, in comparison with normal elderly subjects (Grober & Buschke, 1987; Miller, 1975; Morris et al., 1983). For instance, Snodgrass and Corwin (1988) showed demented patients were impaired on recognition of pictures. Alzheimer patients also show deficits in verbal, figural and spatial recognition memory (Salmon, Granholm, McCullough, Butters, & Grant, 1989). Recognition cues appear to be less effective in facilitating memory performance, than other cues. Morris et al. (1983) found that recognition cues were less effective than were "structural cues" (first three letters of the word) in dementia patients, compared to normal elderly subjects. Despite the low level of performance that is observed in dementia patients, recognition level can be influenced by some task manipulations. For example, Miller (1975) found that demented patients responded better on a recognition task, when each correct word was paired with an incorrect word (i.e., forced-choice recognition), compared to a situation where the total number of words were presented with a similar number of other words in a single display.

In summary, it can be concluded that despite the relative preservation of procedural memory in demented subjects, it is clear that dementia patients have gross disturbances in almost, if not all, aspects of episodic memory. Moreover, semantic memory appears to be impaired as well, although, when intentional searches are not required (e.g., priming), there is evidence that semantic memory function is unimpaired.

Having briefly defined dementia and outlined its prevalence, and the nature of the memory deficits seen in dementia, it is now appropriate to describe the nature of depression, to describe the types of memory deficits observed in depression, and to examine whether these deficits are similar to the deficits observed in patients with Alzheimer's disease.

1.5 NATURE OF DEPRESSION

The depressed person characteristically shows a lack of interest in activities that have normally given them pleasure (DSM-III-R, 1987). A person with depressed mood will usually describe feeling depressed, sad, hopeless and discouraged. The third edition of the American Psychiatric Association Diagnostic and Statistical Manual of Mental Disorders (DSM-III-R)(1987), lists the following criteria which must be present for a major depressive episode to be diagnosed:

A. "At least five of the following symptoms have been present during the same two-week period and represent a change from previous functioning; at least one of the symptoms is (1) depressed mood, or (2) loss of interest or pleasure.

(1) Depressed mood most of the day, nearly every day, as indicated either by subjective account or observation by others.

(2) Markedly diminished interest or pleasure in all, or almost all, activities most of the day, nearly every day (as indicated either by subjective account or by observation by others of apathy most of the time).

(3) Significant weight loss or weight gain when not dieting (e.g., more than 5% of body weight in a month), or decrease or increase in appetite nearly every day.

(4) Insomnia or hypersomnia nearly every day.

(5) Psychomotor agitation or retardation nearly every day (observable by others, not merely subjective feelings of restlessness or being slowed down).

(6) Fatigue or loss of energy nearly every day.

(7) Feelings of worthlessness or excessive or inappropriate guilt (which may be delusional) nearly every day (not merely self-reproach or guilt about being sick).

(8) Diminished ability to think or concentrate, or indecisiveness, nearly every day (either by subjective account or as observed by others).

(9) Recurrent thoughts of death (not just fear of dying), recurrent suicidal ideation without a specific plan, or a suicide attempt or a specific plan for committing suicide.

B. (1) It cannot be established that an organic factor initiated and maintained the disturbance.

(2) The disturbance is not a normal reaction to the death of a loved one.

C. At no time during the disturbance have there been delusions or hallucinations for as long as two weeks in the absence of prominent mood symptoms.

D. Not superimposed on Schizophrenia, Schizophreniform Disorder, Delusional Disorder, or Psychotic Disorder (p.222)."

The DSM-III-R (1987) also provides guidelines for diagnosing the severity of major depression:

In "mild" depression, few, if any, symptoms are present in excess of those required to make the diagnosis, and the symptoms result in only minor impairment or occupational functioning, social activities and relationships with others. In "moderate" depression, the symptoms or functional impairment present fall between "mild" and "severe" indications of depression. In "severe" depression, several symptoms are present in excess of those required to make the diagnosis, and the symptoms markedly interfere with occupational functioning, social activities and relationships with others.

1.5.1 Subclassification of Mood Disorders

According to the DSM-III-R (1987) classifications, mood disorders are divided into depressive disorders (unipolar depression), in which the individual suffers only depressive symptoms without ever experiencing mania; and bipolar disorders (or manic depression), in which both depression and mania occur. Mania is defined by excessive elation, expansiveness, irritability, talkativeness, inflated self-esteem, a greatly reduced need for sleep, grandiose delusions, intense activity, increased sociability, and flight of ideas which involves a continuous flow of accelerated speech which is often disorganised and incoherent (DSM-III-R, 1987, p.215). Frequently, the person does not recognise any change in their behaviour and resists efforts and suggestions for treatment. The existence of mood disorders, which change in apparently opposite directions, has given rise to the name of affective disorders to embrace unipolar depression, bipolar depression, and mania (Rosenhan & Seligman, 1984). Further divisions of bipolar depressive category, include bipolar disorder, which is defined by the presence of manic episodes which require treatment and usually hospitalisation (Willner, 1985); and cyclothymia, in which the person has not had a remission of manic or depressive symptoms for at least two months in duration, over a two year period.

Bipolar depressions are clearly distinguishable from unipolar depression. They involve swings between episodes of mania and episodes of depression. These swings in mood may alternate rapidly over a few days, or occasionally, occur almost simultaneously. There may be rapid shifts of mood to anger or depression, lasting only moments, or hours. The depressive disorders have also been further divided into major depression, in which there is one or more major depressive episodes; and dysthymia, in which depression occurs for most of the day and almost every day, over a period of at least two years. In addition, a current major depressive episode can be specified as melancholic type (or endogenous depression), and this form of depression responds well to somatic antidepressant therapy, such as, tricyclics, lithium, and electroconvulsive shock. Melancholic type major depressive episode is thought to arise from a disordered biochemistry of the brain (Rosenhan & Seligman, 1984).

The majority of research studies into the cognitive deficits associated with depression have focussed upon major depression (or unipolar depression)(Hart, Kwentus, Hamer, et al., 1987; Hart, Kwentus, Taylor, et al., 1987; La Rue, 1989; La Rue, D'Elia, Clark, Spar, & Jarvik, 1986; O'Connor et al., 1990; O'Hara, Heinrichs, Kohout, Wallace, & Lemke, 1986; Weingartner, Cohen, Murphy, Martello, & Gerdt, 1981).

1.5.2 Predisposing Factors to Depression

The rate of onset of a major depressive episode is variable, the symptoms developing over days or weeks. In some cases, however, onset may be abrupt. For example, when associated with severe psychosocial stressful events, such as the loss or death of a loved one, a life-threatening illness, marital separation, or divorce. Other risk factors include major social difficulties, including, retirement, problems with housing, difficulties with family, and poor health (Burvill, 1988), and psychoactive substance dependence, particularly alcohol and cocaine dependence (Cawley, Post, & Whitehead, 1973). Not all people faced with adverse life events become depressed. Murphy (1982) found that those most vulnerable to depression also lacked an intimate supportive relationship, which would have provided partial immunity to adverse life events. Psychological depression may also result from lowered motivation, as described by the arousal-state hypothesis (W.R. Miller, 1975; Weingartner, Miller, & Murphy, 1977), or lack of perceived reinforcement, as described by the learned helplessness model of depression (Seligman, Klein, & Miller, 1975).

Several authors have drawn a distinction between early-onset depression, where the first episode of depression occurred before the age of 60 years, and late-onset depression, in which the first episode occurred after the age of 60 years (Burvill, Stampfer, & Hall, 1986; Hirschfeld et al., 1989). Personality traits in subjects aged between 31 and 41 years, of decreased emotional strength, increased interpersonal dependency, and increased thoughtfulness, were found to be associated with the early-onset of major depression (Hirschfeld et al., 1989). Patients with late-onset depression are said to be significantly less likely to have a family history of affective illness and to have a more stable premorbid personality (Burvill et al., 1986). Prognosis in terms of recovery from depression for those aged 60 years and over, is thought to be poor, and the recovery is believed to be more protracted with increasing age and chronic physical health problems (Murphy, 1983).

1.5.3 Neuroanatomical Basis of Depression

Various methods have been used to assess regional brain dysfunction in depressed patients. Positron emission tomography (PET scanning) allows highly localised assessment of metabolic activity, in the form of glucose metabolism. The assumption is that metabolic activity can be measured by the amount of glucose utilised during a monitored activity. Baxter et al. (1985) and Buchsbaum et al. (1984) have demonstrated that in some experimental conditions, bipolar patients show a global reduction in cerebral metabolic rate for glucose, compared to unipolar depressed patients, and that a subgroup of unipolars may have a specific decrease in left frontal activity. These two studies indicate that possible differences in regional metabolic activity between depressed and control subjects are task dependent.

Scalp electroencephalogram (EEG) recordings can also be used to assess brain function in depressed patients. Perris (1975) studied 28 unipolar and four bipolar depressed patients, and found that the EEG activity over both hemispheres changed with recovery from depression. These changes were more pronounced over the left hemisphere and the more depressed the individual, the greater the change over the left hemisphere relative to that over the right hemisphere. D'Elia and Perris (1973) found predominantly left hemisphere EEG changes in the occipital region of the brain in their study of 18 unipolar depressed patients.

A third method of assessing brain function in depressed patients is to measure regional cerebral blood flow. Sackeim et al. (1990) studied 41 patients with major depressive disorder and found marked reduction in global cortical blood flow, compared to normal control subjects. The reduction was most apparent in the frontal and temporoparietal association areas of both hemispheres. According to the authors, these areas serve arousal, attentional, and motivational functions, and may therefore, be held partially responsible for lowered arousal in depression (Weingartner et al., 1983). At variance with these results is the study by Silfverskiold and Risberg (1989) who found no significant difference between groups on cerebral blood flow during depression and following recovery. However, Wood and Flowers (1988) found that a verbal recognition task revealed deficient performance for bipolar patients tested during the manic and depressive phases of the disorder. Gur and colleagues (1984) reported similar findings for their depressed patients on a verbal analogies test.

These studies offer conflicting results in assessing brain dysfunction in depressed individuals. Under certain experimental conditions, there appears to be a loss of the normal anterior/posterior gradient in metabolic activity (Buchsbaum et al., 1984), whilst under other conditions, some patients have a decrease in left frontal activity (Baxter et al., 1985). In addition, changes in the ratio of left to right hemisphere EEG amplitude has been demonstrated over the occipital regions of the brain, although this change was found to be greater for the left hemisphere (D'Elia & Perris, 1973; Perris, 1975).

Although no consistent pattern has emerged it would seem that some brain alteration accompanies a depressive disorder, and that the specific change may depend upon the nature of the depressive disorder. It is possible that depressed patients suffer a predominately left hemisphere dysfunction and that the frontal and temporoparietal areas are involved.

1.5.4 Biochemical Deficiencies in Depression

Reduced levels of neurotransmitters in the brains of depressed patients have given rise to the hypothesis of a biochemical cause of depression. It has been found that there is a deficiency in catecholamines, particularly cholinergic and dopaminergic systems (Willner, 1985). This deficiency in catecholamines has a shared similarity to the hypothesised biochemical causation of Alzheimer's disease (Rossor, 1982). As already indicated in the section discussing the causes of dementia, the cholinergic system would appear to be particularly important to learning and memory (Caine et al., 1981; Sitaram, Weingartner, & Gillin, 1978).

Another system which may be affected in depression is the noradrenergic system. This system appears to be important in maintaining the level of arousal, which in turn, may play a role in memory performance (Willner, 1985). Conversely, disruption in brain state arousal and activitation due to a noradrenergic deficiency, may be one factor which accounts for encoding failures seen in depressive patients. This hypothesis has been supported by several studies using drug treatments to enhance the arousal-activation state seen in depression. For example, Reus, Silberman, Post, and Weingartner (1979) investigated the drug d-Amphetamine's activation of noradrenergic function and stimulation of arousal on word recall, using free and cued recall tasks. The results showed an increase in verbal free recall only, suggesting specific effects on memory processes. These authors hypothesised that induced levels of arousal prolonged the life of the short-term memory trace and therefore the length of time available for consolidation of material.

L-dopa which facilitates catecholaminergic function, particularly dopamine, has been shown to improve performance in serial and free recall learning tasks in a depressed population (Henry et al., 1973; Murphy, Henry, & Weingartner, 1972), but not in the affective symptomatology of depression (Alexopoulos, 1989). This facilitation in memory has been attributed to an increase in arousal levels produced by the drug.

It would seem that changes in the concentrations of brain monoamines, especially catecholamines, create disturbances in levels of arousal and motivation, which subsequently contributes to the learning and memory impairments observed in depressed patients. Evidence in support of a biochemical basis to the disruption in learning has been forthcoming from several studies. Treatment with drugs which stimulate cholinergic activity, have been observed to enhance learning and memory in depressed patients, however, a similar improvement in affective state, has not been found.

1.6 MEMORY DEFICIT IN DEPRESSION

Depression is associated with a range of deficits in many areas of mental functioning. As with dementia, deficits in memory are often observed. Evidence of difficulty in semantic memory will be discussed first, followed by a discussion of procedural and episodic memory abilities.

1.6.1 Semantic Deficits in Depression

There is little empirical evidence concerning the status of semantic memory function in depressives. The only study which has directly examined semantic abilities in depression was that of La Rue et al. (1986). They found depressed patients performed equally with control subjects on the naming of common objects within given categories, whilst by comparison, demented patients performed poorly at this task. It would appear then that depressives do not display gross deficits in semantic memory.

1.6.2 Procedural Memory Functioning in Depression

Squire, Shimamura, and Graf (1985) found procedural memory intact when investigating memory functioning in depressed patients. Using word completion tests as a priming task (procedural memory test) and a recognition test (explicit, episodic task), depressed patients were found to be severely impaired on the recognition test, but not on the word stem priming task. More recently, Hertel and Hardin (1990) investigated the effects of depressed mood on remembering information. College students who received depressive mood inductions, or who were naturally depressed, showed deficits in recognition (explicit, episodic memory) but not in homophone spelling (procedural memory). In the spelling procedure, subjects were asked a series of questions. Homophones contained in some of the questions were worded to reflect the homophone's less common meaning. The spelling of targets in the less common form indicated that subjects responded to experimental material implicitly to the same degree as nondepressed subjects, even though their explicit memory was inferior to that of nondepressed control subjects.

1.6.3 Episodic Memory Deficits in Depression

Many studies have found impaired explicit memory in depressed individuals (Sternberg & Jarvik, 1976; Stromgren, 1977), although memory impairment is by no means always found (Hart, Kwentus, Hamer, et al., 1987; Henry, Weingartner, & Murphy 1973; O'Connor et al., 1990; Popkin, Gallagher, Thompson, & Moore, 1982; Whitehead, 1973).

Acquisition

Whitehead (1973; 1974) reported that depressed patients showed impairments in synonym learning relative to controls, or to baseline conditions. Henry et al. (1973) reported that depressed patients showed deficits in performance of secondary (long-term memory) tasks, such as impaired serial learning on the second and subsequent trials, but not on the first trial, and Gibson (1981) reported poorer performance on free recall tasks. In other depressed patients impairments have been observed on verbal paired associate learning tasks, and on backward digit span (Cohen, Weingartner, Smallberg, Pickar, & Murphy, 1982; Stromgren, 1977; Whitehead, 1973). Depressed patients are more likely to demonstrate impairments in memory for learning nonorganised, in comparison to semantically organised, word lists (Weingartner, Cohen, et al., 1981), and when using a selective reminding procedure for verbal learning (Buschke, 1973; Hart, Kwentus, Hamer, et al., 1987), recalling fewer words without reminding, compared to the control subjects.

In contrast, Kendrick and Post (1967), O'Hara et al. (1986), O'Connor et al. (1990), Popkin et al. (1982), Sternberg and Jarvik (1976) and Whitehead (1973; 1974) have no impairments in paired associate learning in their respective studies looking at memory complaints and performance in the depressed elderly.

Possible reasons for the discrepant findings in the above studies may be indicative of sampling differences, based upon the severity of depressive symptoms. For example, La Rue (1989), Roy-Byrne, Weingartner, Bierer, Thompson & Post (1986) and Weingartner, Cohen, et al. (1981) used patients hospitalised for their disorder, whilst Hart, Kwentus, Hamer, et al. (1987) used outpatients tested prior to drug treatment. There is also a discrepancy in the severity of depression of subjects used in the various studies, for example, Roy-Byrne et al.'s depressed sample were moderate to severely depressed, Weingartner, Cohen, et al.'s patients were moderately depressed, and mildly depressed subjects were used in the Hart, Kwentus, Hamer, et al. study. A number of studies have reported that the severity of memory impairments is correlated with the severity of depression (Cohen et al., 1982; Henry et al., 1973; Sternberg & Jarvik, 1976; Stromgren, 1977).

It is clear that deficits in acquisition are most evident in tasks which require sustained concentration. Processing conditions that require the use of more elaborate encoding operations for storing information in memory has been described by Hasher and Zacks (1979) as "effortful" processing. Weakly processed or less "effortful" processing conditions, such as acoustic processing, remain unaffected in depression, whilst operations requiring sustained concentration and greater efficiency of information processing appear to be impaired (Weingartner, Cohen, et al., 1981). Consistent with this position it has been shown that when depressed subjects were asked to impose organisation on a list of random words, a task requiring greater effort, depressives demonstrated significant impairments. Weingartner, Cohen, et al. (1981) found that depressed patients were, however, able to take advantage of structure inherent in a word list, particularly if that structure was organised. Access to previous knowledge (semantic memory) is available, in the depressed patient, if given sufficient time to complete the task (Weingartner, 1984; Williams & Scott, 1988).

Contradictory findings however, have also been reported. Levy and Maxwell (1968) varied the structure of material presented to each subject in the form of work lists, by varying the levels of approximation-to-text. Their results found that the depressed group benefited less than control subjects from increasing structure. The control subjects also demonstrated increased word recall with increasing approximation-to-text, whilst the depressed group did not show the same linear trend.

To explore this apparent inconsistency, Watts, Dalgleish, Bourke, and Healy (1990) presented subjects with both semantic category word lists and lists approximating-to-text. The highest level of structure comprised complete clustering of words into their specific categories, the medium level provided partial clustering, whilst at the lowest level of structure, words were presented in a random order. Three levels of approximation-to-text were also presented. When high and medium levels of approximation-to-text were compared, less memory impairment was evident on highly structured materials, for the depressed group. This was interpreted by Watts and colleagues as being, in part, a perception by the depressed subject, of difficulty with the unstructured material and of a reduction in attentional resources in depression, making the effort required to organise unstructured material, too demanding. No significant interaction was found between depression and levels of structure for semantic category word lists, in contrast to the Weingartner, Cohen et al. (1981) results.

Thus, there is still no consensus as to the extent, or nature, of the acquisition deficit in depression.

Storage

A normal rate of forgetting learned material has been observed in depressed subjects for recognition of line drawings, as assessed by Hart, Kwentus, Taylor, et al. (1987), althought the depressed patients required a longer exposure time (4 vs. 2 seconds) to learn the equivalent number of drawings, relative to control subjects. This seems to suggest that while it may take longer for depressed subjects to acquire information, they demonstrate a normal forgetting rate.

Retrieval

Depressed patients are more likely to demonstrate impairments in free recall in comparison to cued recall tasks (Cohen et al., 1982), and in comparison to recognition tasks (Hart, Kwentus, Taylor, et al., 1987; La Rue, 1989; Roy-Byrne, et al., 1986; O'Connor et al., 1990; Stromgren, 1977).

In a review of the literature concerning recognition, a task requiring less "effort", Hasher and Zacks (1979) concluded that depressives show less deficits on recognition compared to free recall tasks. Subsequently, Hart, Kwentus, Hamer, et al. (1987) found that depressed patients performed as control subjects on a verbal recognition memory task. Some contradictory evidence that recognition performance is unimpaired in depressives, has come from other studies. For example, Wood and Flowers (1988) found impairment for depressed patients, relative to healthy subjects, on a verbal recognition task. Further, Gibson and Kendrick (1976) and Gibson (1981) found verbal and nonverbal recognition memory loss in elderly depressed patients. Cole and Zarit (1984) and Sternberg and Jarvik (1976) also found deficits in recognition memory in a group of depressed hospitalised patients. However, Miller and Lewis (1977), using signal detection analysis of verbal recognition memory deficits, showed that depressed patients were less willing to guess when uncertain. This suggests that, while recognition memory may be impaired in depressed subjects, the impairment may reflect the patients' willingness to make errors rather than an impairment in memory capacity or retrieval.

To summarise, to this point, the problems in differential diagnosis have been described, and the characteristics of dementia and depression have been outlined. As already indicated, the difficulties in differential diagnosis would be mitigated if dementia and depression were characterised by different types of cognitive deficit (that is, if these deficits differed qualitatively from each other, and from those deficits observed in normal elderly subjects). This brief review of the deficits in dementia and depression has yielded a number of possible differences in memory capacity. At the same time, both disorders are associated with intact procedural memory, so tests of implicit memory are unlikely to provide a basis for differentiation. Dementia, but not depression, is associated with impairments in semantic memory, so prima facie, differentiation might be achieved by using semantic tasks, such as word naming. However, because semantic memory deficits occur much later in the disease process than episodic deficits, it is unlikely that semantic memory tasks will provide sensitive differentiation at that early stage when diagnosis is most difficult. Most research into the nature of the difference in deficits between dementia and depression has concentrated on qualitative differences in episodic memory performances.

1.7 THEORIES ABOUT THE BASIS FOR DIFFERENCES IN MEMORY DEFICITS

Several researchers have directed their energies towards isolating cognitive impairments exclusive to the demented and depressed patient (Cohen et al., 1982; Coghlan & Hollows, 1984; Gibson, 1981; Hart, Kwentus, Hamer, et al., 1987; Hart, Kwentus, Taylor, et al., 1987; La Rue, 1989; La Rue et al., 1986; Reus et al., 1979; O'Connor et al., 1990; Weingartner, Cohen, et al., 1981; Weingartner et al., 1977; Weingartner et al., 1981), in an attempt to differentiate Alzheimer's disease from depression in the elderly. Other theorists have attempted to develop models of the type of deficits associated with dementia and depression, although their aim has not been specifically to provide a basis for clinical differentiation of the two disorders (Caine et al., 1981; Cermak, 1979; Craik, & Lockhart, 1972; Craik & Tulving, 1975; Drachman & Leavitt, 1974; Hasher & Zacks, 1979; Kintsch, 1970; Lewis, 1979; W.R.

Miller, 1975; Moscovitch et al., 1986; Shimamura et al., 1987; Simon, 1976; Tulving & Thomson, 1973; Weingartner, 1985; Wickelgren, 1973).

Although there have been a number of suggestions about the type of deficits that might distinguish dementia from depression, by far the most popular, and most researched, is that made by Weingartner et al. (1981). On the basis of memory performance with categorised and noncategorised word lists, Weingartner claimed that demented patients were unable to use the structure of the material they were learning to organise their memories, while depressed patients (Weingartner, Cohen, et al., 1981) were able to use organised structure for list learning. Weingartner considered that demented subjects experienced difficulty accessing the semantic knowledge necessary to appreciate and encode ongoing events and stimuli. The encoding of Alzheimer patients was thus considered less meaningful and elaborate than that of normals, leading to defective episodic memory. Weingartner claimed, however, that the memory deficits of depressed subjects were not explained by a failure in semantic encoding, but were better described in terms of the generalised impaired ability to perform effortful, but not automatic, memory operations (Weingartner, 1985). These failures in effortful processing have been said to arise from motivational deficits associated with depression (Hart, Kwentus, Hamer, et al., 1987). The distinction between effortful and automatic processes will be briefly elaborated here so that deficits observed in depression can be evaluated, with reference to it, in the following sections. Following this, the Weingartner model will be critically reviewed.

1.7.1 Effortful Versus Automatic Processes

Effortful processes are defined by Hasher and Zacks (1979) as processes that require effort and thereby limit one's ability to engage simultaneously in other effortful processes. Tasks which are considered "effortful" are those which *require* the subject to organise and elaborate material at the time of encoding. Effortful processes are ones which require sustained attention, and are more easily disrupted than less consciously active operations. Tasks which require encoding strategies are more sensitive to declines in effortful processing. An example of an effortful task is the learning of a list of unrelated words, since, in order for the task to be well performed, encoding strategies are required. In contrast, automatic operations are defined in part by their capacity to be carried on simultaneously with other tasks with little cost to their performance. Tasks which are considered "noneffortful" or automatic are those which can be accomplished without focussed attention, and which require little cognitive capacity. Automatic processes, because of their minimal drain on capacity, should not be significantly affected by altered cognitive capacity, because sustained attention to accomplish the task is not required. They are said to function at a constant level under all circumstances, including stressful and fatigue situations (Hasher & Zacks, 1979). An example of a task which is less effortful than learning a list of unrelated words would be one where the list of words is already organised, such as learning a list of semantically, clustered words. Recognition is also regarded as less effortful than recall because it is considered to require less effort at the time of retrieval. Also, organisation of learning materials and the development of retrieval strategies play larger roles in recall than in recognition. In a recall task, the information about the item stored in semantic memory must be complete, otherwise the item cannot be reconstructed. This is not required for successful recognition (Simon, 1976).

1.7.2 Evidence in Favour of Weingartner's View that Episodic Deficits of Dementia Patients are due to Difficulty in using Semantic Knowledge

Weingartner's conclusion that dementia patients have difficulty accessing the semantic knowledge necessary to encode ongoing events was based on several lines of evidence. First, Weingartner et al. (1981) gave subjects two lists of words for recall. One list was categorised, the other list was not. Whilst normal subjects recalled more words from the related than unrelated list, dementia patients did not. The performance of the demented patients was not facilitated by the presence of semantic structure (i.e., categories in the list). Second, the clustering in dementia patients recall performance did not improve, as normal subjects did, across trials, and was not greater as normal subjects was, for related compared to unrelated words. This suggested that Alzheimer patients were not imposing any organisation on the related word list. Weingartner attributed these results to an inability to access structures in semantic memory, thereby leading to inadequate or weak encoding of material.

Indirect evidence for Weingartner's model has come from other sources. First, as noted above, semantic memory deficits, such as impaired word naming, knowledge of semantic attributes, and category membership (Bayles, 1982; Butters et al., 1987; Ober et al., 1986), are common deficits observed in dementia. If there were a loss of semantic information, or a breakdown of access to such information, as has been proposed by some theorists (Martin & Fedio, 1983), then Weingartner's hypothesis that dementia patients are incapable of appreciating the semantic *properties* of the material is fully consistent with the semantic memory evidence.

Further evidence for the inability of dementia patients to use semantic information in memory encoding comes from the work of Davis and Mumford (1984).

They showed that demented patients failed to show a differential improvement when presented with category cues in comparison to free recall. According to Davis and Mumford, the result could be explained by assuming that information has not been processed and hence encoded according to its semantic properties, and therefore, could not be used as a retrieval aid.

However, despite the general support given to Weingartner's hypothesis, there are a number of criticisms that can be made. These criticisms relate to (1) methodological aspects of Weingartner's study; (2) alternative theoretical accounts of the observed deficits (Buschke, 1984; Miller, 1975); (3) studies which have found facilitation of learning by semantic organisation (Cushman et al., 1988; Nebes et al., 1989); and, (4) studies showing that retrieval deficits are also important (Morris et al., 1983; Tuokko & Crockett, 1989).

Methodological and theoretical criticisms of Weingartner's hypothesis

The first criticism of Weingartner's study is that a "floor" effect may have obscured the differential advantage for categorised words. Because the performance of the dementia patients was very low, it is possible that dementia patients did appreciate the semantic information and used this knowledge to encode the information, but that the effect was obscured. Thus, Weingartner's hypothesis would be made more convincing if a similar specific deficit were found when tasks less subject to "floor" effects, such as recognition or cued recall tests were used. As noted by many researchers, a chief difficulty in experimental studies of memory in Alzheimer's disease lies in the inherent high level of difficulty and demand for sustained attention of a standard free recall task. Consequently, some researchers (e.g., Cushman et al., 1988; Morris et al., 1983; Nebes et al., 1989) have chosen to focus on tasks which reduce the amount of conscious effort required by a subject yet still allow examination of active encoding and retrieval processes. In this way the ability of the demented patient to utilise, in any way, a given strategy in memory functioning (e.g., categorisation), can be more carefully assessed and compared with possible decreased efficiency in using such a strategy. An example of a memory task which reduces the overall cognitive demand on subjects is cued recall, which in fact provides a more thorough assessment of items in storage than does free recall (Tulving & Pearlstone, 1966).

Alternative theoretical accounts of observed deficits

A second problem with Weingartner's account is that alternative theoretical accounts may equally explain the findings. Weingartner claimed that his patients were able to sort exemplars into categories, even though they were unable to use the information in encoding. It may therefore, be the case that demented patients are able to effectively encode semantic information, but only to the extent that their encoding is induced and directed by the stimulus material itself, and is not an intentional act (Nebes et al., 1989). The deficit in dementia may be more globally described as a deficit in "voluntary" processing. This broader account may explain the deficits in semantic memory when intentional memory search is required, as well as those of episodic tasks.

Studies which have found facilitation of learning by semantic organisation

A third criticism, related to the second point, is that there is evidence that dementia patients can encode information semantically under some circumstances. Studies which suggest dementia patients may encode information semantically, include the research by Cushman et al. (1988) and Nebes et al. (1989). In contrast to the Weingartner et al. (1981) findings, Cushman et al. (1988) found that dementia patients did show better recall with related than unrelated word lists. Although the magnitude of the significant effect was small, Cushman suggested that this result indicated that demented patients were still sensitive, albeit to a limited degree, to the differences in semantic properties. Further evidence that dementia patients do retain some knowledge of semantic associations has come from the Nebes et al. (1986) and Nebes and Brady (1988) studies. These researchers found that at least some components of semantic memory and function have been spared in dementia, when the learning is incidental. Further, using primary tasks on word naming and lexical decision tasks, Nebes et al. (1989) found that the demented group showed a significantly larger priming effect than the control group, suggesting that greater benefit was gained by the dementia patients use of this method.

Studies in support of a retrieval deficit in dementia

A fourth criticism of Weingartner's conclusion is that studies of retrieval strategies suggest that the memory deficit in dementia may not be one exclusively of encoding, but also one of retrieval (Buschke, 1984; Morris et al., 1983; Miller, 1975). The cued recall paradigm has been utilised to assess storage and retention on the basis that free recall, alone, may not recover all items available in storage (Tulving & Pearlstone, 1966). Miller (1975) and Morris et al. (1983) employed a cued recall paradigm with demented patients. Word recall improved significantly when the initial letter of each cue word was presented at the time of recall, whilst free recall and recognition were significantly impaired. The evidence regarding the effectiveness of semantic cues in retrieving information is much less clear cut. As noted earlier, semantic cues appear to be less effective that structural cues in facilitating retrieval. Yet, the findings of Buschke (1984) and Cushman and colleagues (1988), suggest that some semantic information may be encoded at the time of learning, in that category cue recall did improve performance. Further support for the retrieval deficit explanation comes from the work of Tuokko and Crockett (1989) who studied free and cued recall in demented patients. They suggested that the retrieval deficits were more pronounced than were acquisition deficits in mildly demented patients.

In summary, although Weingartner et al.'s (1981) views about the nature of the semantic deficit in dementia have some support, criticisms can also be made of the methodology used to demonstrate the specific deficit. Moreover, there are alternative theoretical explanations which may account for the findings to date.

1.7.3 Evidence for the View that the Episodic Deficits in Depressed Patients are due to Deficits in "Effortful" Processing

The conclusion by Weingartner, Cohen, et al. (1981) that memory deficits in depression could not be explained by a failure in semantic encoding, but were better described in terms of a generalised impaired ability to perform "effortful" memory operations, was based on a number of lines of evidence. First, depressed subjects show greater impairments on tests of free recall than on tests of recognition (La Rue et al., 1986), and on tests of free recall compared to incidental learning (Roy-Byrne et al., 1986). Secondly, impairment was greater for highly unrelated items than highly related ones (Weingartner, Cohen, et al., 1981). The most interesting finding was that depressed patients benefited more than normal subjects from the organisation of words into categorised lists (Weingartner, Cohen, et al., 1981).

Weingartner, Cohen, et al. (1981) used three experiments to demonstrate how encoding strategies might influence the later recall of information in depressed patients. The depressed subjects were required to learn word lists which differed in amount of structure or organisation. Weingartner employed related/categorised, related/unclustered, and unrelated word lists. The depressed patients demonstrated memory failures when asked to impose their own organisation on the related/unclustered, and unrelated word lists, compared to control subjects. However, unlike the demented patients in their later study (Weingartner et al., 1981), the depressed group did not differ from control subjects in recall after processing word lists that were highly organised and in which the organisation was shown by obvious clustering. From these results, Weingartner argued that the more effort required to encode a task into long-term memory, the greater will be the impairment for the depressed patient. It is of importance to note that Weingartner's sample of depressed subjects was not matched in age to the demented group in his later study. In addition, the rate at which the list of words were read to the depressed subjects, was not the same as that used for his later study using demented patients.

1.7.4 Criticisms that can be made of Weingartner's View about the Deficits in Depression

Criticisms of the methodology and interpretation can be made of Weingartner, Cohen, et al.'s (1981) study of depressed patients.

First, the depressed subjects differed markedly in age from the demented subjects in Weingartner's original research. The mean age of depressed subjects was 44.2 years, whilst dementia patients had a mean age of 61.2 years. The importance of comparing similar age range subjects is emphasised given the knowledge that cerebral functioning declines after the age of 60 years (Lezak, 1983).

Secondly, Hertel and Hardin (1990) have recently reviewed recognition memory deficits associated with depression and claimed that deficits are associated with a loss of spontaneous use of strategies. Hertel and Hardin interpreted the recognition memory deficits as being due to a deficit in the initiation of strategies beneficial to the task. On tasks that were well structured with detailed instructions on how to initiate the recognition task, depressed subjects performed on a level which was equivalent to that for control subjects. Hertel and Hardin (1990) findings contrast with those of Hasher and Zacks (1979), who viewed memory deficits in the depressed patient as being due to a reduction in the cognitive capacity available to complete an "effortful" type task.

1.8 AIMS AND HYPOTHESES

The study to be reported in this thesis examined the learning-memory processing deficits of mildly demented and depressed patients and compared their performance to healthy elderly control subjects, focussing particularly upon the retrieval conditions that facilitate the recall of related and unrelated word lists.

1.8.1 Rationale

To date, there is some support for Weingartner's ideas about the nature of the memory deficits in depression and Alzheimer's disease. However, part of the difficulty in evaluating Weingartner's hypothesis is that a free recall task was used to evaluate encoding, whereas recognition or cued recall may be more appropriate to the demented patient. In addition, *since there has been no attempt to date to directly examine demented and depressed subjects on similar test stimuli in the same study, the conclusions from Weingartner's studies are somewhat questionable, particularly as his patient groups were from different age groups.* Ideally, learning and memory performance by both elderly demented and elderly depressed groups, should be compared with each other and with the results for healthy elderly control subjects within the same study.

1.8.2 Hypotheses

On the basis of Weingartner's theory of processing failures in dementia and depression, a number of predictions can be made about the expected performance of demented, depressed and control groups relative to each other on categorised and noncategorised word lists, and in recall, cued recall and recognition conditions.

The hypotheses regarding encoding processes and retrieval conditions are discussed separately.

Encoding Processes

Figure 1 schematically illustrates the predicted performance pattern of the demented, depressed and control groups for the learning-memory tasks with related and unrelated words, based upon Weingartner's hypotheses and findings (Weingartner, Cohen et al., 1981; and Weingartner et al., 1981). Related lists of words are lists containing words which are examples of the same category group. Unrelated word lists contain words which are not categorisable. Figure 2 schematically illustrates the predicted performance of all three groups in the recall of related words in the form of semantic clusters for each learning trial.

According to Weingartner's model, the demented subjects, due to an inability to gain access to semantic structures, and hence an inability to encode new material efficiently, should:

(a) recall significantly fewer related and unrelated words in comparison to depressed and control subjects;

(b) recall no more related than unrelated words;

(c) show no, or very little, acquisition across learning trials; and

(d) show no clustering of words into categories at the time of recall. Normal subjects will often recall words in clusters, i.e., will repeat one list item immediately after another list item from the same semantic category. For example, they will recall the word "apple" followed immediately by recall of the word "orange".

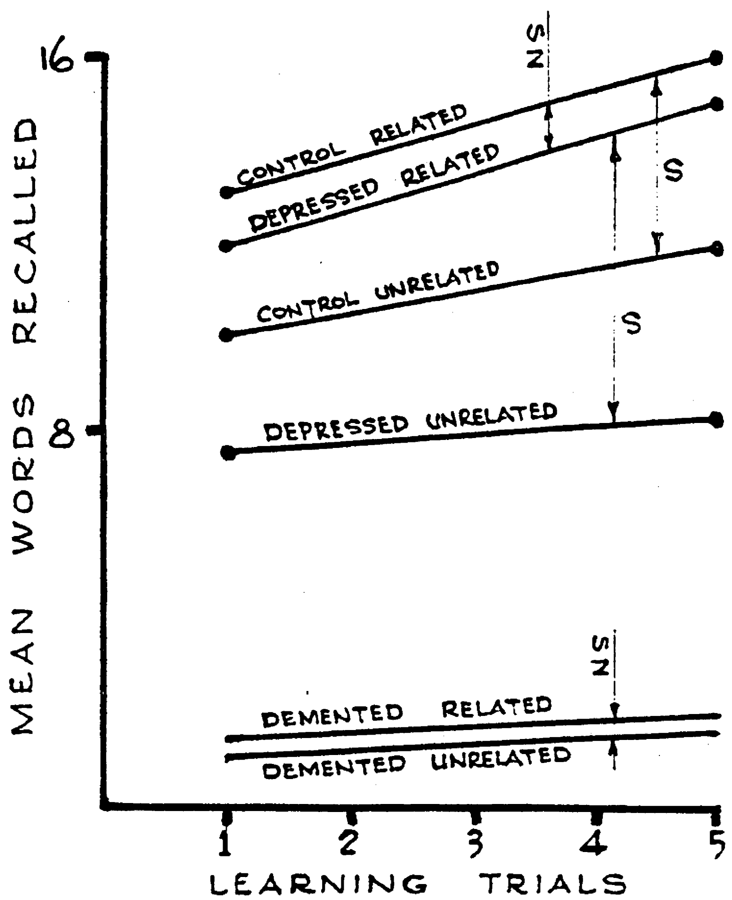

Figure 1: Schematic drawing of predicted performance by each group, for related and unrelated word lists across five learning trials.

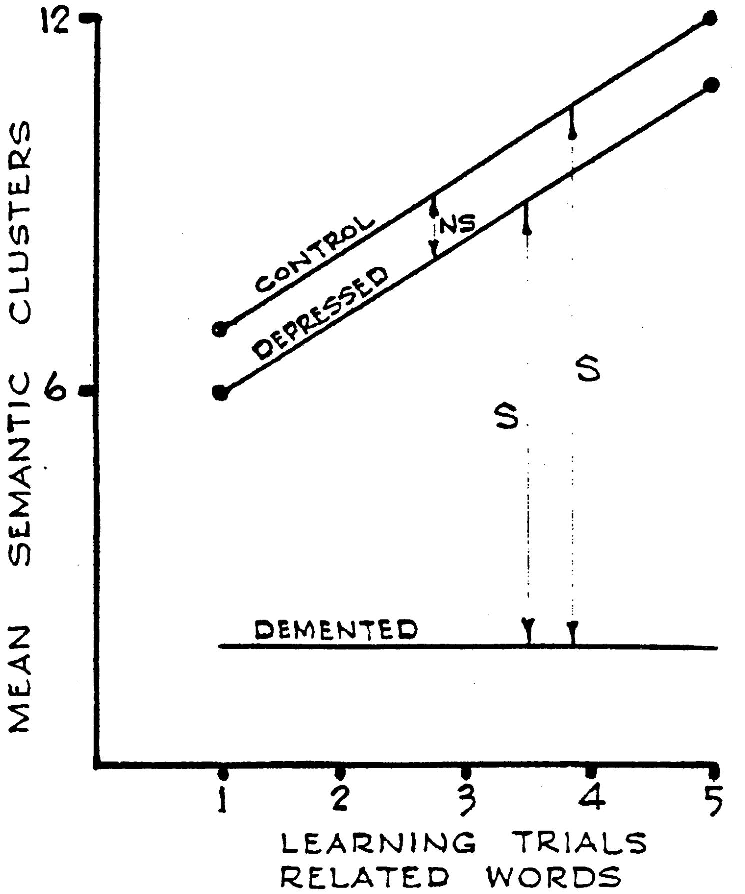

Figure 2: Schematic drawing of predicted performance by each group on the mean number of words recalled as semantic clusters, over five trials for related words.

It is hypothesised from Weingartner's model that the depressed subjects, because of their retained ability to appreciate and utilise semantically structured material, should:

(a) not differ from the control subjects in the number of related words recalled across learning trials, nor will they differ in the number of related words recalled in the form of semantic clusters from the control subjects. Since this material is already organised, their deficits will not be evident, because no effort is expended in organising it.

(b) However, because of their relative inability to sustain the effort required to impose organisational structure upon a list of random words, the depressed subjects will recall significantly fewer unrelated words than the control subjects.

(c) The depressed and control subjects will demonstrate learning across trials for related and unrelated words, and will recall significantly more related than unrelated words. (In this respect they will differ from demented subjects.)

(d) Because of the depressed patients' hypothesised reduced cognitive capacity to cope with "effortful" tasks, in comparison to the control subjects, the difference between learning across trials for the related and unrelated words for the depressed subjects, will be greater than the difference between the rate of learning related and unrelated words across trials, by the control subjects. (Refer to schematic drawing of predicted results for depressed and control groups. Figure 1.)

Retrieval conditions

Figure 3 schematically illustrates the predicted performance patterns of the demented, depressed and control groups on the three retrieval processes of short-delay free recall, cued recall and recognition, based upon Weingartner's model (Weingartner, Cohen, et al., 1981; and Weingartner et al., 1981). If Weingartner's hypothesis is correct, and encoding is almost entirely impaired in dementia, then dementia patients will fail to benefit from cues at retrieval. If however, a retrieval impairment is largely responsible for the deficit, then the retrieval conditions of cued recall and recognition should facilitate recall. Although retrieval and encoding processes are inextricably linked and interdependent (Tulving, 1983), it is still claimed that information which has not been encoded cannot be retrieved under any conditions. It should be noted here that even if dementia patients are unable to encode any material, they may still achieve a recognition performance level of 50 percent correct. That is, given the forced-choice paradigm recognition task presented, which in this case represents eight words out of a possible 16 words, by chance alone, they may nominate 50 percent of targets. Given that the patients are able to recall a number of items on the free recall task, their recognition performance should be above chance level (i.e., a total of more than eight words must be recognised). Facilitation of memory by recognition cues, then, will only be demonstrated if the number of words recognised is above a level expected on the basis of their recall.

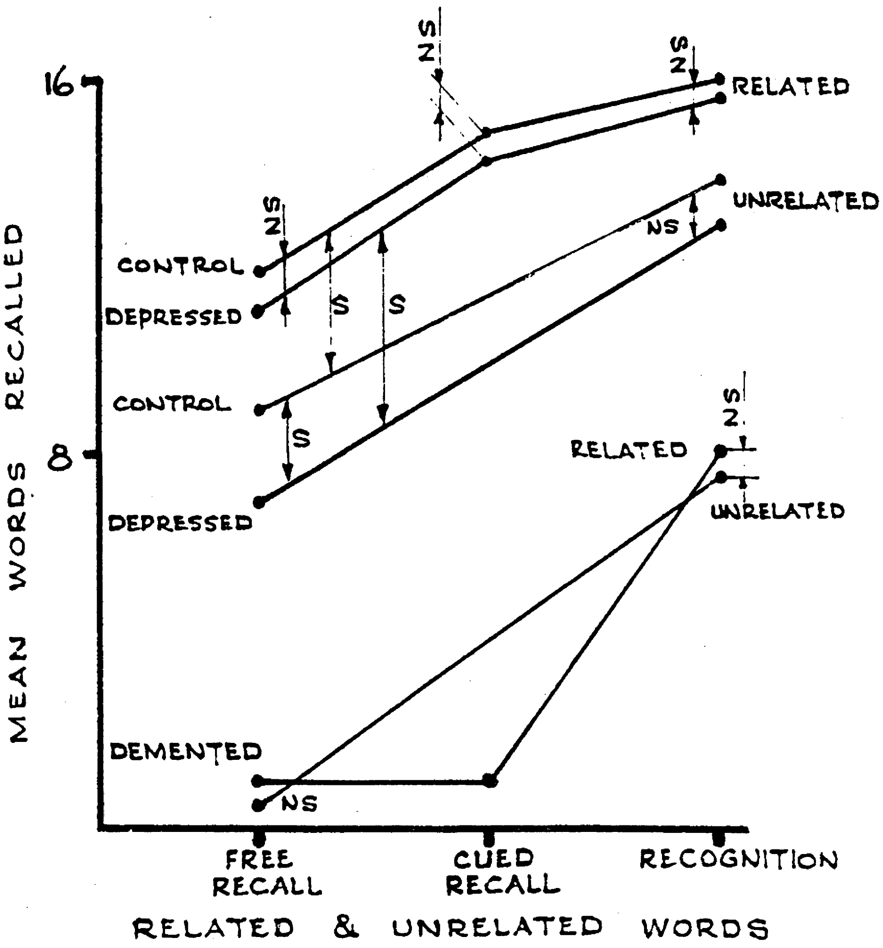

Figure 3: Schematic drawing of predicted results on related and unrelated word lists for each group.

It is hypothesised from Weingartner's model that the demented subjects, because of an impairment in semantic encoding, should:

(a) not be aided by semantic cues in a cued recall task;

(b) show impaired retention following all three retrieval conditions of free recall, cued recall, and recognition, compared to that of depressed and normal subjects;

(c) recall no more related than unrelated words; and,

(d) show not much greater than chance level performance on the recognition task.

It is hypothesised that the depressed subjects, because of their sensitivity to the amount and form of structure present in a word list, should:

(a) show no differentiation on the free recall and cued recall tasks of related words from the control subjects. However, because of their relative poorer performance on "effortful" tasks, the depressed subjects, should:

(b) show significant differences in recall of related and unrelated words, relative to cued recall and recognition, and will recall significantly fewer unrelated words than the control subjects. Because the depressed and control subjects are able to encode information about the meaning of words, semantic properties of the words may serve as cues at retrieval. Thus,

(c) depressed and control subjects will show greater facilitation following cued compared to free recall, and following recognition compared to free-and-cued recall conditions. In addition, as depressed subjects perform essentially as normal subjects on less "effortful" tasks, such as recognition,

(d) depressed and control subjects will show no difference in the number of words recognised on either the related or the unrelated word lists.

CHAPTER TWO - METHOD

2.1 MEASURES

Three measures were administered in screening potential subjects for this study. These consisted of measures of premorbid intelligence, mood, and cognitive functioning.

2.1.1 National Adult Reading Test

Premorbid level of intelligence was measured by the National Adult Reading Test (NART) (Nelson & O'Connell, 1978) (Appendix A). The NART provides a valid estimate of premorbid intelligence since it is known that reading ability is highly correlated with intelligence in the general population (O'Carroll, 1987), and that reading ability of the demented subjects is unimpaired compared with matched controls, at least until the late stages of the disorder (Nelson & McKenna, 1975). A mean Intelligence Quotient (IQ) of between 90 to 109 is defined as an average performance for an adult on the Weschler Adult Intelligence Scale-Revised (WAIS-R) (Wechsler, 1981). An equivalent predicted IQ performance on the NART would be a score of between 23 and 46 errors out of a possible 50 errors.

2.1.2 Geriatric Depression Scale

The Geriatric Depression Scale (GDS) (Yesavage et al., 1983) provides an assessment of mood in elderly subjects, covering material related to cognitive complaints, motivation, future/past orientation, self-image, losses, agitation, obsessive traits, and mood (Appendix B). Of the 30 questions on the GDS, 20 indicate the presence of depression when answered positively while 10 others indicate depression when answered negatively. Based on studies of the GDS (Brink et al., 1982; Yesavage et al., 1983), a score of 11 or greater is considered to be a possible indicator of depression, with a score of 15-21 indicating mild depression, and a score of 22 or greater being indicative of severe depression.

2.1.3 Mini-Mental State Examination

A measure of cognitive status was obtained in all three groups by administering the Mini-Mental State Examination (MMSE) (Folstein, Folstein, & McHugh, 1975) DIS version (Eaton, Regier, Locke, & Taube, 1981) (Appendix C). The Diagnostic Interview Schedule (DIS) is one version of the MMSE which has been widely used in research chiefly due to its brevity and ease of administration, making it especially practical for use in clinical settings. The MMSE permits comparison of cognitive impairment across studies. More sophisticated measures of cognitive impairment would have been too time consuming and were not warranted given the aims of the study. This version of the MMSE is scored out of 30 and comprises 11 items. The MMSE concentrates on the cognitive aspects of functioning, and excludes questions on mood, abnormal mental experiences, and the form of thinking. It is divided into two sections, the first of which requires vocal responses only and covers orientation, short-term memory, and attention and concentration. maximum score on this section is 21. The second part tests ability to name, follow verbal and written commands, write a sentence spontaneously, and copy a complex polygon similar to a Bender-Gestalt Figure (visuospatial construction). The maximum score for this section in 9, making a total maximum score of the MMSE of 30. Most of the publications on the MMSE (Fillenbaum, Hughes, Heyman, George, & Blazer, 1988; Jorm, Scott, Henderson, & Kay, 1988; Teng, Chui, Schneider, & Metzger, 1987), have recommended the cut-off scores of 18-23 correct to indicate mild/moderate cognitive impairments, and 0-17 correct to indicate severe cognitive deficit.

2.2 SUBJECTS

Eight demented patients, eight depressed subjects and five healthy elderly subjects participated in the study. Three were male and 18 were female.

2.2.1 Inclusion Criteria

Subjects were required to be of average premorbid intelligence as assessed by the National Adult Reading Test (NART), to have no history of alcohol abuse, stroke, epilepsy, or cardiovascular disease, or other neurological or psychiatric disorders, other than dementia and depression, and to be healthy, and over 60 years of age. Subjects who may have been suffering from multi-infarct dementia, Korsakoff syndrome, or depression of organic origin, such as infectious or toxic factors, were thus excluded. In addition, subjects from the three groups, were to be Australian born, with English as their first language. Permission to interview the depressed and demented subjects in this study was sought from both the subject and his or her family.

The demented patients were required to meet the diagnostic criteria for "mild" dementia of the Alzheimer type, as outlined in the Diagnostic and Statistical Manual of Mental Disorders-Revised (DSM-III-R) (1987), as diagnosed by a registered psychiatrist. In addition, to be included in the study, patients were required to score between 18 and 23 on the MMSE, which indicated mild cognitive impairment. To avoid the possibility of depression co-occurring in the dementia patients, subjects were excluded if they scored in the moderate-to-severe range of the Geriatric Depression Scale, that is, if they obtained a score over 21 (see Measures section below). Patients were excluded from the study if they presented with profound deafness or greatly impaired vision, as diagnosed by the subject's doctor or optician.

The depressed patients were required to meet DSM-III-R (1987) diagnostic criteria for moderate-to-severe major depressive episode as diagnosed by a psychiatrist. Inclusion to the study also required that subjects score 15 or over on the Geriatric Depression Scale, a score which indicates mild to severe depression (see Measures section below).

The control subjects were required to demonstrate normal cognitive functioning as measured by the Mini-Mental State Examination, that is, to obtain a score of over 23, and normal mood as measured by the Geriatric Depression Scale, by obtaining a score of under 11.

Description of Subjects

The demented subjects (eight females) functioned sufficiently well to remain at home in the care of their respective spouses. They ranged in age from 65 to 84 years. The depressed subjects (two males and six females) were drawn from the psychiatric wards of either Woden Valley Hospital, Canberra, or Prince Henry Hospital, Sydney, and ranged in age from 60 to 85 years. The patients had been depressed for four months to two years prior to hospitalisation, and had been hospitalised for one to six weeks. Half the depressed group had received electro-convulsive therapy (ECT) during their present period of hospitalisation. All the depressed group were receiving anti-depressant medication in the form of tricylic drugs. The five control subjects (one male and four females) resided at the Goodwin Homes for the Aged in the Farrer and Ainslie suburbs of Canberra of the Australian Capital Territory. These five subjects ranged in age from 63 to 89 years.

2.3 GROUP CHARACTERISTICS

Table 1 presents the mean and standard deviation scores on the MMSE, NART and the GDS, together with age and educational level, for all groups. ONEWAY analysis of variance indicated no significant differences between the groups in years of education, $F(2,18) = 1.06$, $p<.36$, age, $F(2,18) = 1.83$, $p<.19$, or level of intelligence as measured by the NART scores, $F(2,18) = 1.49$, $p<.25$. However, a highly significant difference emerged on the MMSE test, $F(2,18) = 20.19$, $p<.000$ and the GDS test, $F(2,18) = 25.02$, $p<.000$. On assessment with the Geriatric Depression Scale the depressed group scored within the moderately-severely depressed range, according to group norms (Brink et al., 1982; and Yesavage et al., 1983). On ONEWAY analysis of variance with planned contrasts, using Tukey's procedure ($p<.05$), the depressed group were significantly more depressed, as measured by the GDS, than the demented and control groups, whilst the demented group emerged significantly more impaired in cognitive functioning, as measured by the MMSE, than the control and depressed groups. The demented group averaged 19.87 on the MMSE (SD: 2.10), indicative of mild cognitive impairment. These data demonstrate that the group selection emerged as intended.

The variable SEX, because of the predominance of female subjects, was also examined using Pearson Product-Moment Correlation (Table 13). Depressive symptomatology as measured by the GDS was found to be more severe for the male subjects as compared to female subjects ($p = .003$).

Table 1

<u>Characteristics of the Control, Depressed and Demented Groups</u>

Charactistic Number (n)	Control Group n=5		Depressed Group n=8		Demented Group n=8	
	Mean	(SD.)	Mean	(SD.)	Mean	(SD.)
Age	76.00	(11.76)	69.00	(9.60)	77.37	(6.65)
Education (years)	13.60	(3.34)	13.00	(0.00)	13.75	(1.38)
MMSE*	27.80	(0.83)	24.87	(2.94)	19.87	(2.10)
NART (errors)	17.00	(3.80)	22.25	(6.67)	23.00	(7.29)
Predicted FSIQ	114		110		109	
GDS*	6.00	(1.30)	22.12	(5.33)	11.62	(2.61)

Significant differences between groups by ONEWAY analysis of variance are indicated as follows:

*p<.000

2.4 EXPERIMENTAL STIMULI

Weingartner used word lists of between 20 and 32 words for his studies in dementia and depression. Since studies have shown that the average recall for subjects aged 60 years on a list of nine words is approximately five words (Lezak, 1983), the number of words used by Weingartner was probably excessive, since he was attempting to differentiate dementia patients from normal elderly subjects. The Rey Auditory Verbal Learning Test (Rey, 1964), which consists of 15 words, is used to measure immediate memory span and reveal learning strategies with brain damaged patients (Lezak, 1983). It seemed appropriate to use a word list in accordance with measures well established in clinical practice. Thus, two word lists, each consisting of 16 words were chosen for the present study.

2.4.1 Related Word Lists

Subjects were required to learn a list of 16 high frequency related words over five learning trials, drawn from Kucera and Francis' (1967) word frequency analysis of present-day American English words. Each word had a high degree of association to one of four category names drawn from Battig and Montague (1969) (Appendix D). Each word was no longer than six letters and all words were nouns. The related word list consisted of four words each from four categories, making a total of 16 words altogether. Each category consisted of high frequency words from the categories "Fruit", "Clothing", "Parts of the Body", and "Animals", and were selected from Battig & Montague's (1969) category norms. The words were arranged in a clustered form and the order of the clusters was varied systematically (Appendix E). Thus, List A contained the following words, in the following order: apple, orange, banana, pear, shirt, socks, pants, coat, legs, arms, head, eye, dog, cat, horse, cow. Following the five learning trials, subjects were given an interference test, consisting of 16 unrelated words. The interference list (List B), contained the following words in this order: desk, ranger, house, moon, stove, mountain, glasses, river, bell, boat, coffee, gun, pencil, church, garden, colour (Appendix F) and were selected from the Rey AVLT (Rey, 1964), Lists A and B. All words selected for the interference list were free from any semantic relationship with the related/clustered words of List A, using the Battig and Montague (1969) category norms for verbal items. Following the presentation of List B, the cued recall and recognition tasks were given. The recognition trial comprised all words from the related word list, together with a list of new words. The list of 16 new words were all nouns, and were matched on word frequency and word length with the original words (Appendix I).

2.4.2 Unrelated Word List

The unrelated words consisted of items matched in word frequency to the categorised words taken from Kucera and Francis (1967). The order of the words was varied systematically (Appendix J). Thus, List A, the unrelated word list, contained the following words in the following order: cheese, farmer, chisel, robe, cloud, glen, comic, weapon, chair, radio, water, game, judge, craft, price, tea (Appendix D). List B, the interference list for unrelated words, contained the following words in this order: weather, parent, rose, soap, school, nest, calendar, typewriter, lock, candle, fight, knife, box, jar, medal, tunnel (Appendix F), and were also selected from the Rey AVLT (1965), Lists A and B. All words selected were free from any semantic relationship to List A's words, related and unrelated word lists, using the Battig and Montague (1969) category norms for verbal items. The recognition trial comprised all words from the unrelated word list, together with a list of new words. The list of 16 new words were all nouns, and were matched on word frequency and word length with the original words (Appendix L).

2.5 PROCEDURE

For subjects from each group, the experimenter read out the words at a rate of one word every two seconds, and subjects recalled as many as they could immediately following the end of the list. This procedure differs slightly from the Weingartner studies, where words were read to the depressed group every two seconds, whilst the demented patients were presented words every three seconds. The number of correct responses, perseverations (repeated items), intrusions (nonlist items), and semantic clusters were scored. The maximum cluster score was 12 for each trial. Thus, if a subject recalled all items of the semantic categories sequentially, the maximum points for semantic clusters were awarded. Following the five learning trials, an interference trial of 16 words was commenced (Appendix F), and an immediate free recall requested. A short-delay recall test of the related words was then requested, and subjects' responses noted. A cued-recall test was then administered for the related word conditions. The subject was provided each of the four category names of the semantic categories used in the related word list (Appendix G). For example, "from the first list I read out to you, can you remember any words that were animals?" A recognition trial then followed (Appendix H). The paired items in the recognition test were presented in a random order, in a forced choice format, with the examiner reading aloud each of the paired words. For example, "Is the word you have heard before, apple or mask?" Subjects were required to verbally identify the word which they had heard previously. The number of correct words identified were scored.

In the case of the unrelated word condition, each subject learnt a list of unrelated words having the same frequency of occurrence in the English language as the related words presented previously (Appendix J). This list of unrelated words was presented over five learning trials. The procedure for unrelated words was identical to that outlined for the related/clustered word list, with the exception that a cued-recall test was not administered (because no semantic categories were present, it was inappropriate to cue word responses with category names). A recognition test then followed (Appendix K). The procedure was identical to that outlined for the recognition test of related words. The presentation of the two different word lists was separated by one day with the order of presentation counterbalanced across subjects within a group. Half the subjects learnt the related words first and half were given the unrelated words to learn prior to the related word list.

The list procedure was similar to that used in the administration of the California Verbal Learning Test (CVLT) (Delis, Kramer, Kaplan, Fridlund, & Ober, 1987), but it is important to describe how the current procedure differed from that of the CVLT.

The CVLT is a measure of verbal learning and memory for adults, including the elderly. The test measures how learning occurs, or fails to occur, as well as the amount of verbal material learned. The subject is required to learn a list of 16 words (four words in each of four semantic categories) over five trials. The categories are: "fruits", "spices" and "herbs", tools", and "clothing". An interference list (List B) of 16 words is then presented for one trial. List B comprises four 4-item categories, of which two categories overlap with List A (fruits, spices and herbs), and two are different from List A (fish, kitchen utensils). This latter feature of List B is included to observe if semantically similar items to those in List A cause greater interference than nonsimilar items. Immediately after free recall of List B, a free recall of List A is requested. A cued-recall trial follows, where the subject is provided each of the four semantic categories to facilitate recall of List A items. These free-and-cued-recall trials of List A comprise the "short-delay" trials. After a 20 minute delay, the "long-delay" trials comprising free recall, cued recall, and recognition testing for List A is requested. The number of correct responses, perseverations (repeated items), intrusions (nonlist Items), and semantic clusters are scored for each trial.

The present procedure differed from the California Verbal Learning Test (CVLT) (Delis et al., 1987) in the following ways:

(1) The CVLT does not include an unrelated word list.

(2) Words used as stimuli, in the current study, were all chosen from Kucera and Francis (1967) frequency norms of present-day American English, with no word being longer than six letters and all words being nouns. In addition, degree of association between word frequency and category name were drawn from Battig and Montague (1969). Delis and his colleagues (1987) did not specify from which source they drew their selection of words, and neither did they specify word-length, type of word used, or the degree of association to category name.

(3) The recognition trial also represented a modification of the CVLT procedure. Within the CVLT procedure, the recognition trial involves the presentation of 44 items, 16 from List A and 28 distractor words. There are five types of distractor words: four List B items from semantically similar categories to List A, four List B items from semantically dissimilar categories, four items not previously presented from semantically dissimilar categories to List A, and eight items with phonological similarities to individual List A items. The subject is required to say "yes" if an item was from List A and "no" if it was not. In the present study, the recognition procedure differed to the following extent: the number of words from which the subject was required to select the correct word was reduced to 32 words. In addition, the distractors are drawn from a completely new list of words, matched in word frequency, word-length and word-type to the related or unrelated word lists.

(4) The CVLT includes a 20 minute delay before the free recall test, followed by cued recall and recognition tasks. The present study omitted this long delay recall task. Immediate free recall was required after each of the five learning trials. A short delay followed with the interference list (List B), which was then followed by free recall, cued recall and recognition tasks.

To summarise, each subject was given two lists of words to recall across five learning trials, followed by an interference trial. Retention was measured by recall and recognition procedures following the interference trial. A cued recall test was also used for the related word list. The free-and-cued-recall trials of the related word list comprise the "short-delay" trials of the CVLT.

CHAPTER THREE – RESULTS

3.1 ANALYSES

A univariate split-plot analysis of variance design was applied to examine the data, using the SPSS-X MANOVA programme (1988). There was one between-subjects variable (groups) and two within-subject variables (type of words and learning trials). Tests of simple main effects were then employed, conditional upon the univariate analysis rejecting the null hypothesis of no interaction.

Means and standard deviations for the control, depressed and demented groups for all tasks are presented in Table 4. Individual test results for the control, depressed and demented groups are provided in Tables 5, 6 and 7.

3.2 ACQUISITION PHASE

3.2.1 Word Recall

The number of related and unrelated words recalled over the five learning trials by all groups was examined in the first analysis. The mean words recalled for each group, each list type, and for each trial are presented in Figure 4. Reference may also be made to Table 2 for means and standard deviations. The ANOVA (see Table 3) yielded a significant main effect for group, $F(2,18) = 22.37$, $p<.000$, type of words, $F(1,18) = 97.62$, $p<.000$, and trials, $F(4,72) = 27.83$, $p<.000$. In broad terms these findings indicated differences in the level of performance between groups, that more related than unrelated words were recalled, and that the number of words recalled increased across trials.

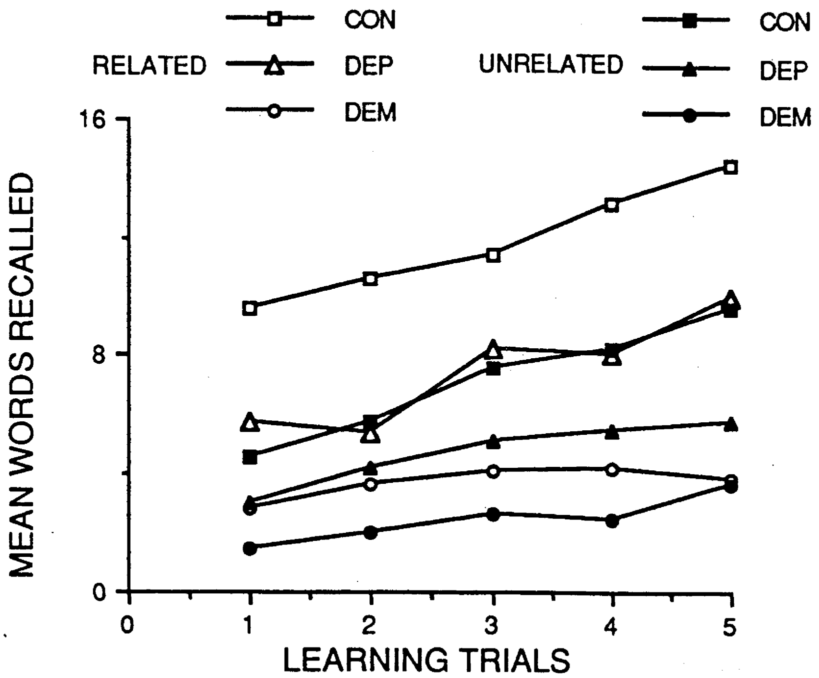

Figure 4: Mean words recalled over five trials for each group on related and unrelated word lists.

Table 2

Mean Words Recalled of Control, Depressed and Demented Groups on Each of the Five Learning Trials of Related and Unrelated Words

	Control Group		Depressed Group		Demented Group	
Trial	Mean	(SD.)	Mean	(SD.)	Mean	(SD.)
Related Words						
1	9.60	(3.64)	5.75	(2.31)	2.87	(2.53)
2	10.60	(3.36)	5.37	(2.50)	3.62	(1.59)
3	11.40	(3.05)	8.25	(2.31)	4.12	(1.72)
4	13.20	(2.58)	8.00	(3.11)	4.25	(1.58)
5	14.40	(2.07)	10.00	(3.81)	3.87	(1.45)
Unrelated Words						
1	4.60	(1.34)	3.00	(1.92)	1.50	(1.06)
2	5.80	(0.83)	4.25	(1.38)	2.00	(1.30)
3	7.60	(1.14)	5.12	(2.74)	2.62	(1.59)
4	8.20	(1.92)	5.50	(2.67)	2.50	(1.60)
5	9.60	(3.05)	5.75	(3.05)	3.62	(1.92)

Table 3

Analysis of Variance Summary Table of Demented, Depressed and Control Groups with Related versus Unrelated Words and Learning Trials

	SS	df	MS	F
Between Subjects				
groups	1274.67	2	637.33	22.37*
Subjects within groups	512.80	18	28.49	
Within Subjects	78.07	18	4.34	
words	423.41	1	423.41	97.62*
groups by words	88.16	2	44.08	10.16**
trials	280.70	4	70.17	27.83*
groups by trials	54.41	8	6.80	2.70***
words by trials	2.49	4	0.62	0.26
groups by words by trials	25.65	8	3.21	1.37

* p<.000 ** p<.001 *** p<.01

There was no significant three-way interaction (Groups x Words x Trials), nor was there a significant interaction between type of words and trials, which indicated that the rate at which related and unrelated words were learnt, did not differ. However, significant interaction effects for trials by group, $F(8,72) = 2.70$, $p<.012$, and type of word by group, $F(2,18) = 10.16$ $p<.001$, which indicated that learning across trials occurred at a different rate for the groups and that the difference between the recall of related words and unrelated words was different for the groups.

In order to identify the source of significant interaction effects, the data were further examined by conducting analyses of simple main effects (Appendix M). Each group was examined separately for variation in performance across trials for related and unrelated words. Both the control and depressed groups showed significant learning across trials (controls: $F[4,16] = 13.22$, $p<.000$; depressed: $F[4,28] = 13.09$, $p<.000$. However, the demented group's learning effect just failed to attain significance $F(4,28) = 2.67$, $p=.053$. It had been predicted that the depressed group would recall proportionately more related than unrelated words compared to the control group (see schematic drawing of predicted results Figure 1, Aims and Hypotheses section). The predicted effect was not found. The depressed group's performance on the related word list was poorer than predicted (control: $F[4,16] = 13.22$, $p<.000$, depressed: $F[4,28] = 13.09$, $p<.000$).

It had also been predicted that the dementia patients would fail to take advantage of semantic organisation. In contrast to the prediction, the demented group together with control and depressed subjects, learnt related words better than unrelated words (control: $F[1,4] = 33.27$, $p<.004$; depressed: $F[1,7] = 58.98$, $p<.000$; demented: $F[1,7] = 8.70$, $p<.021$). However, the difference between related words and unrelated words was greater for the control and depressed groups than for the demented group, a result consistent with the prediction.

Table 4

Mean Words Recalled of Control, Depressed and Demented Groups
on Learning-Memory Tasks of Related and Unrelated Word Lists

Task	Control Group		Depressed Group		Demented Group	
	Mean	(SD.)	Mean	(SD.)	Mean	(SD.)
Related Words	14.40	(2.07)	10.00	(3.81)	3.87	(1.45)
Semantic Clusters	10.40	(2.07)	6.50	(3.02)	2.12	(1.12)
Free Recall	12.60	(4.15)	7.75	(3.88)	1.12	(1.35)
Cued Recall	14.00	(1.58)	10.62	(3.77)	9.37	(2.13)
Recognition	16.00	(0.00)	15.62	(0.51)	12.12	(2.64)
Unrelated Words	9.60	(3.05)	5.75	(3.05)	3.62	(1.92)
Free Recall	7.00	(4.00)	3.37	(3.02)	1.12	(1.80)
Recognition	15.40	(0.89)	15.25	(0.88)	10.62	(1.50)

Table 5: Learning-Memory Test Information from Five Healthy Elderly Controls

		CONTROL SUBJECTS								
SUBJECTS:		**1**	**2**	**3**	**4**	**5**	**MEAN**	**SD**	**RANGE**	**BEST POSSIBLE SCORE**
INFORMATION AND TESTS	AGE:	70	88	70	89	63	76.00	11.76	63-89	
	MMSE:	29	28	28	27	27	27.80	0.83	27-29	30
	SEX:	M	F	F	F	F				
IRREGULAR WORD READING (ERRORS)	NART:	20	13	15	15	22	17.00	3.80	13-22	50 (errors)
DEPRESSION SCALE	GDS:	10	10	1	7	2	6.00	1.30	1-10	30
RELATED WORDS:		15	16	14	11	16	14.40	2.07	11-16	16
SEMANTIC CLUSTERS:		11	12	10	7	12	10.40	2.07	7-12	12
FREE RECALL:		15	16	15	6	11	12.60	4.15	6-16	16
SEMANTIC CLUSTERS:		11	12	11	4	8	9.20	3.21	4-12	12
CUED RECALL:		15	16	14	12	13	14.00	1.58	12-16	16
RECOGNITION:		16	16	16	16	16	16.00	0.00	16	16
UNRELATED WORDS:		13	7	12	6	10	9.60	3.05	6-13	16
FREE RECALL:		12	4	9	2	8	7.00	4.00	2-12	16
RECOGNITION:		16	14	15	16	16	15.40	0.89	14-16	16
YRS. EDUCATION:		13	13	16	13	13	13.60	1.34	13-16	

Table 6: Learning-Memory Test Information from Eight Depressed Patients

PATIENT:		1	2	3*	4	5*	6*	7	8*	MEAN	SD	RANGE	BEST POSSIBLE SCORE
		DEPRESSED PATIENTS											
INFORMATION	AGE:	61	80	85	63	63	65	75	60	69.00	9.60	60-85	
AND	MMSE:	29	21	23	23	25	29	23	26	24.87	2.94	21-29	30
TESTS	SEX:	M	M	F	F	F	F	F	F				
IRREGULAR WORD READING (ERRORS)	NART:	25	30	24	26	10	15	27	21	22.25	6.67	10-30	50 (errors)
DEPRESSION SCALE	GDS:	29[c]	26[c]	16[b]	29[c]	16[b]	22[c]	19[b]	20[b]	22.12[c]	5.33	16-29	30
RELATED WORDS:		11	5	5	11	8	13	11	16	10.00	3.81	5-16	16
SEMANTIC CLUSTERS:		8	3	2	7	5	9	7	11	6.50	3.02	2-11	12
FREE RECALL:		11	2	7	8	5	7	7	15	7.75	3.88	2-15	16
SEMANTIC CLUSTERS:		7	1	5	5	1	4	3	11	4.62	3.29	1-11	12
CUED RECALL:		12	2	11	12	10	11	12	15	10.62	3.77	2-15	16
RECOGNITION:		16	15	16	15	15	16	16	16	15.62	0.51	15-16	16
UNRELATED WORDS:		5	2	3	8	3	7	7	11	5.75	3.05	2-11	16
FREE RECALL:		5	0	0	5	2	6	1	8	3.37	3.02	0-8	16
RECOGNITION:		16	14	14	16	15	16	15	16	15.25	0.88	14-16	16
YRS. EDUCATION:		13	13	13	13	13	13	13	13	13.00	0.00	13	

* ECT patients
b = moderately depressed c = severely depressed

Table 7: Learning-Memory Test Information from Eight Patients with Dementia of the Alzheimer Type

PATIENT:		1	2	3	4	5	6	7	8	MEAN	SD	RANGE	BEST POSSIBLE SCORE
		DEMENTIA PATIENTS											
INFORMATION AND TESTS	AGE:	65	80	84	78	74	84	72	82	77.37	6.65	65-84	
	MMSE:	18	20	19	23	18	20	23	18	19.87	2.10	18-23	30
	SEX:	F	F	F	F	F	F	F	F				
IRREGULAR WORD READING (ERRORS)	NART:	28	11	21	15	24	25	34	26	23.00	7.29	11-34	50 (errors)
DEPRESSION SCALE	GDS:	13[a]	12[a]	12[a]	9	15[b]	11[a]	7	14[a]	11.62[a]	2.61	9-15	30
RELATED WORDS:		2	4	2	5	4	5	3	6	3.87	1.45	2-6	16
SEMANTIC CLUSTERS:		1	3	1	3	2	1	2	4	2.12	1.12	1-4	12
FREE RECALL:		0	2	0	0	3	1	3	0	1.12	1.35	0-3	16
SEMANTIC CLUSTERS:		0	1	0	0	0	0	2	0	0.37	0.74	0-2	12
CUED RECALL:		10	9	10	8	5	11	10	12	9.37	2.13	5-12	16
RECOGNITION:		10	2	10	14	8	16	14	13	12.12	2.64	10-16	16
UNRELATED WORDS:		6	3	4	6	3	4	0	3	3.62	1.92	0-6	16
FREE RECALL:		0	0	0	0	4	4	0	1	1.12	1.80	0-4	16
RECOGNITION:		9	11	10	12	12	11	12	8	10.62	1.50	8-12	16
YRS. EDUCATION:		13	13	16	16	13	13	13	13	13.75	1.38	13-16	

a = possible indication of depression   b = moderately depressed

3.2.2 Semantic clustering of Related Words Across Learning Trials

A second main ANOVA examined for differences between the groups in recalling words on each trial in the form of semantic clusters. As indicated previously, a semantic cluster was scored whenever the subject recalled one list word immediately after another list word from the same semantic category. The maximum cluster score was 12 for each trial. The analysis yielded a significant main effect for group, $F(2,18) = 25.95$, $p<.000$, and for trials, $F(4,72) = 9.14$, $p<.000$, indicating that there were differences in performance level between groups, and that the frequency of clustering increased across trials. There was a significant interaction between trials and group, $F(8,72) = 2.73$, $p<.011$, which indicated that the groups differed in the degree to which semantic clustering increased across trials. Mean semantic clusters for each group over the five trials are presented graphically in Figure 5. A summary table of the ANOVA on all groups for semantic clusters is provided at Table 9. Reference may also be made to Table 8 for means and standard deviations.

Table 8

Mean Words Recalled in the Form of Semantic Clusters of Control, Depressed and Demented Groups on Each of the Five Learning Trials of Related Words

	Control Group		Depressed Group		Demented Group	
Trial	Mean	(SD.)	Mean	(SD.)	Mean	(SD.)
1	6.40	(2.88)	3.37	(1.06)	1.87	(1.64)
2	7.00	(2.64)	3.00	(1.60)	2.37	(1.30)
3	8.40	(2.40)	4.37	(2.64)	2.37	(1.50)
4	9.20	(2.77)	5.37	(2.38)	2.00	(1.06)
5	10.40	(2.07)	6.50	(3.02)	2.12	(1.12)

Table 9

Univariate Analysis of Variance Procedure for Repeated Measures Summary Table of Demented, Depressed and Control Groups with Related Words in the form of Semantic Clusters

	SS	df	MS	F
Between Subjects groups	578.11	2	289.06	25.95*
Subjects within groups	200.52	18	11.14	
Within Subjects	161.01	72	2.24	
trials	81.79	4	20.45	9.14*
groups by trials	48.81	8	6.10	2.73**

* p<.000 ** p<.01

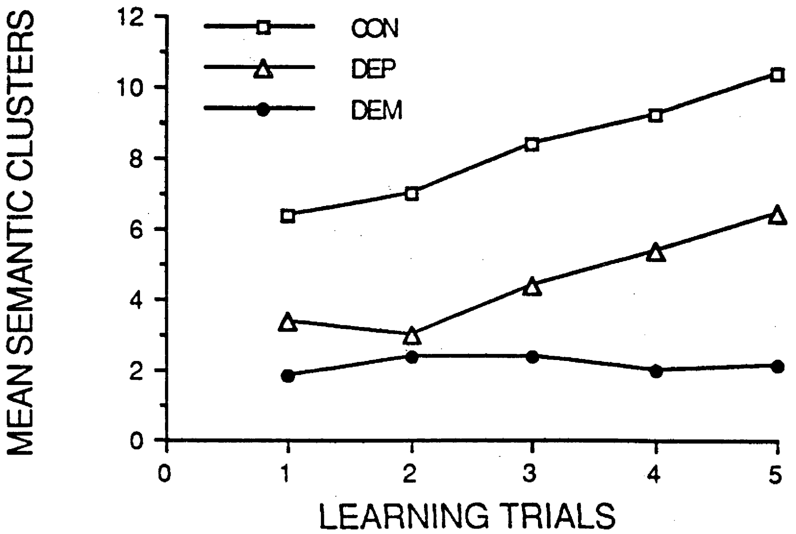

Figure 5: Mean semantic clusters across trials for each group, on the related word lists.

The data were further examined by conducting an analysis of simple main effects (Appendix N). In recalling words across the five learning trials, the control and depressed groups showed a significant increase in the number of semantic clusters reported with repeated presentations of each trial (control: $F[4,16] = 4.45$, $p<.013$; depressed: $F[4,28] = 7.21$, $p<.000$). The demented group showed no significant variation in the number of semantic cluster scores reported over the five trials ($p<.921$). These results are consistent with the predicted pattern of performance (see schematic drawing of predicted results Figure 2, Aims and Hypotheses section). It should be noted that there is a relationship between the number of semantic clusters and the number of words recalled, such that the number of semantic clusters increases as the number of words recalled increases. Because demented subjects had such poor recall performance, it is difficult to judge the proportion of those words which were recalled in clusters, thus, making these findings of semantic clustering difficult to interpret.

3.2.3 Intrusions and Perseverations

A Kruskal-Wallis one-way analysis of variance procedure for non parametric tests was employed to compare the groups separately for intrusive and perseverative words cumulated across trials. A non-parametric test was selected, since the present data did not satisfy distribution assumptions necessary for parametric testing. Intrusions were defined as nonlist word responses and perseverations were defined as repetitions of responses previously given on the same trial. The Chi-square value was corrected for ties throughout these analyses. Because overall scores were too low to retain separate scores for each of the five learning trials, mean intrusions and perseverations with standard deviations were obtained by cumulating across trials to attain a single measure for each group, and each list type. These are shown in Table 10.

Table 10

Mean Level of Intrusions and Perseverations on Related and Unrelated Words for the Control, Depressed and Demented Groups

Error Type	Control Group			Depressed Group			Demented Group		
	Mean	(SD.)	Range	Mean	(SD.)	Range	Mean	(SD.)	Range
RELATED WORDS									
Intrusions	0.60	(1.34)	0-2	0.00	(0.00)	0	1.87	(1.64)	0-2
Perseverations	2.80	(4.08)	0-8	1.12	(0.83)	0-2	0.50	(0.75)	0-2
UNRELATED WORDS									
Intrusions	2.00	(2.00)	0-2	0.62	(0.74)	0-2	0.27	(0.74)	0-1
Perseverations	2.80	(3.70)	0-4	0.12	(0.35)	0-1	0.00	(0.00)	0

Intrusions

Variation in the number of intrusions across learning trials was examined for the related and unrelated word lists, for each group separately. Following the Kruskal-Wallis tests, significant variation was observed across groups in the total number of intrusions produced on the related words (Chi-square = 7.322, $p<.025$). The demented group produced more intrusions than the control and depressed groups. Each of these intrusions was a member of a category from which the list items were drawn. For example, if the words "coat", "vest", and "pants" were recalled, "vest" is an intrusive item, or nonlist word, and is semantically similar to the other words. No significant difference in the number of intrusions on the unrelated word list was observed amongst the groups ($p<.124$).

Perseverations

Kruskal-Wallis one-way analyses of variance examined variations in the number of perseverations for the three groups across learning trials of related and unrelated words. This analysis yielded a nonsignificant effect for all groups on related words ($p<.187$), indicating that no one group produced significantly more perseverations than another group. However, a significant effect was found for unrelated words (Chi-square = 7.951, $p<.018$), with the control group producing significantly more perseverations than the depressed and demented groups (control: 14, depressed: 9, demented: 4). All perseverations for all the groups were from the word lists. For example, these words were recalled in the following order: "shirt", "coat", "socks", "orange", "apple", "banana", "pear", coat", and "shirt". The last two words represent perseverations of the first two correct items recalled.

3.3 RETRIEVAL PHASE

3.3.1 Related Words

A third ANOVA was used to examine performance on the free recall, cued recall and recognition trials for the related word list. The analysis revealed significant main effects for group, $F(2,18) = 17.90$, $p<.000$, and retrieval conditions, $F(2,36) = 61.18$, $p<.000$, and a significant interaction between retrieval conditions and group, $F(4,36) = 7.23$, $p<.000$. This result indicated that there were differences in the level of performance between groups, and the type of retrieval conditions yielded different results for the groups. Mean scores and standard deviations for retrieval conditions are presented in Figure 6. The summary tables for the ANOVA is presented in Table 11.

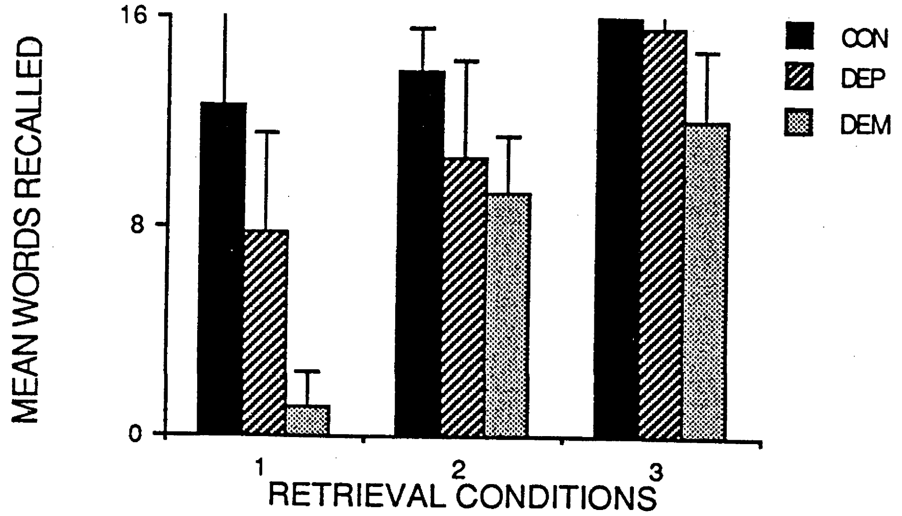

Figure 6: Mean scores and standard deviations for free recall, cued recall and recognition tasks, related words, for each group.

Table 11

Univariate Analysis of Variance Summary Table of Demented, Depressed and Control Groups with Retrieval Processes for Related Words

	SS	df	MS	F
Between Subjects groups	431.74	2	215.87	17.90*
Subjects within A	217.03	18	12.06	
Within Subjects	163.05	36	4.53	
retrieval	554.16	2	277.08	61.18*
groups by retrieval	131.05	4	32.76	7.23*

* $p<.000$

Simple main effects analysis was employed to compare the retrieval condition for each group separately (Appendix M). For control subjects, the performance across the retrieval conditions did not differ ($p<.097$). Recognition performances were almost perfect, with normal subjects performing at ceiling level. The demented group recalled significantly more words on the cued recall and recognition tasks, than they did on the free recall task, $F(2,14) = 65.15$, $p<.000$, indicating that as the task became less effortful, more words were recalled. The depressed group, as expected, showed significant increases in word retrieval in the cued and recognition conditions, $F(2,14) = 25.44$, $p<.000$.

As expected, the demented group had poorer memory performance on all retrieval conditions compared to control subjects. Depressed subjects achieved a similar score on the cued recall and recognition tasks to the control group (depressed cued recall: M = 10.62, recognition: M = 15.62, control cued recall: M = 14.00, recognition: M = 16.00), whilst demented patients attained significantly fewer correct words on free recall and recognition tasks than the depressed group (demented free recall: M = 1.12, recognition: M = 12.12, depressed free recall: M = 7.75, recognition: M = 15.62). Results inconsistent with predictions were that:

(a) significant differences between depressed and control subjects occurred on the free recall task for related words, and

(b) no differentiation of demented and depressed groups was apparent on the cued recall task (see schematic drawing of predicted results Figure 3, Aims and Hypotheses section).

As expected, for all three groups greatest facilitation retrieval was on recognition of words. If all groups were operating at chance level, we would expect a recognition score of eight words for each group. The demented group retrieved 12 out of a possible 16 words on the recognition task. This represents an above chance level of performance by the demented group on the recognition task ($p<.03$). In contrast to Weingartner's prediction, demented patients also benefited greatly by the addition of a semantic cue. The depressed group, as expected, showed an increase in word recall when cues were given. The control group's improvement between the cued recall and recognition retrieval processes (cued recall: M = 14.00, recognition: M = 16.00), was not significant. However, as previously indicated, the control group demonstrated a ceiling effect in the recognition of related words.

3.3.2 Unrelated Words

The fourth ANOVA was performed for all three groups with repeated measures on free recall and recognition retrieval conditions. This analysis yielded significant main effects for group, $F(2,18) = 15.38$, $p<.000$, and retrieval conditions, $F(1,18) = 285.81$, $p<.000$. A significant main effect for group indicated that the groups differed on the number of words recalled, whilst a main effect for retrieval conditions indicated that the number of words recalled under the two variables of free recall and recognition, were different. A nonsignificant interaction effect for group and retrieval conditions ($p<.071$), indicated that the status of the group did not influence the pattern of words recalled under the two retrieval conditions. That is, the pattern of words recalled on the free recall and recognition tasks did not differ significantly for all groups. Mean scores and standard deviations for retrieval conditions are presented graphically in Figure 7. The summary table of the ANOVA analysis is presented at Table 12. Refer also to Table 4.

Table 12

Univariate Analysis of Variance Procedure for Repeated Measures Summary Table of Demented, Depressed and Control Groups with Retrieval Processes for Unrelated Words

	SS	df	MS	F
Between Subjects				
groups	193.22	2	96.81	15.38*
Subjects within A	113.29	18	6.29	
Within Subjects	62.04	18	3.45	
retrieval	985.06	1	985.06	285.81*
groups by retrieval	21.25	2	3.08	0.07

* $p<.000$

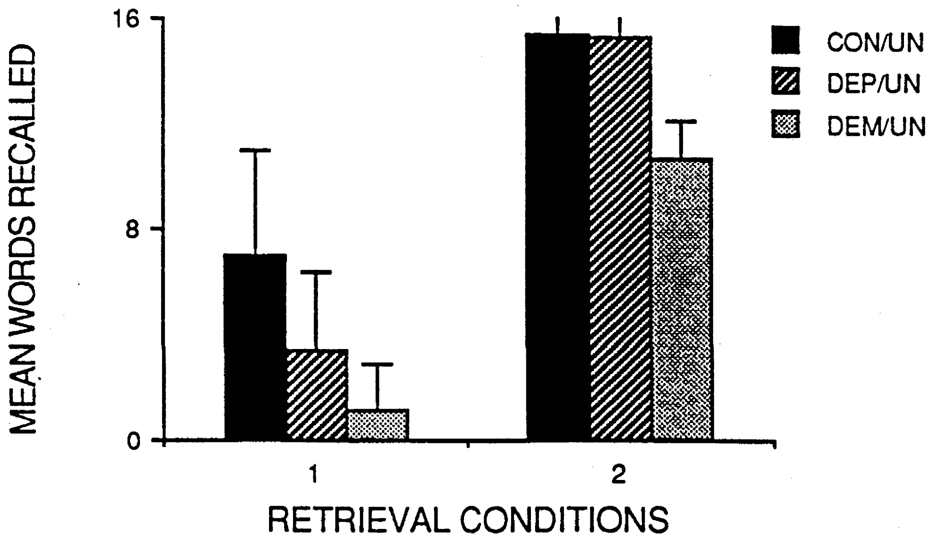

Figure 7: Mean scores and standard deviations for free recall and recognition tasks, unrelated words, for each group.

Table 13

Pearson Product-Moment Correlations for Combined Data on the Variable SEX

	SEX
Group	p = .141
MMSE	p = .155
NART	p = .093
GDS *	p = .003
Related Words	p = .220
Free Recall	p = .153
Cued Recall	p = .321
Recognition	p = .158
Unrelated Words	p = .238
Free Recall	p = .098
Recognition *	p = .044

* Significant

Table 14

Tukey's (HSD) procedure (range at p<.05) for the Depressed Group.

with ECT and without ECT treatment on Memory Tasks

	No ECT		ECT	
Task	Mean	(SD.)	Mean	(SD.)
NART	27.00	(2.16)	17.50	(6.24)
AGE	69.75	(9.21)	68.25	(11.35)
MMSE	24.00	(3.46)	25.75	(2.50)
GDS *	23.75	(4.71)	18.50	(3.00)
Related Words	35.00	(11.43)	39.75	(15.19)
Free Recall	7.00	(3.74)	8.50	(4.43)
Cued Recall	9.50	(5.00)	11.75	(2.21)
Recognition	15.50	(0.57)	15.75	(0.50)
Unrelated Words	21.50	(7.93)	25.75	(12.52)
Free Recall	2.75	(2.63)	4.00	(3.65)
Recognition	15.25	(0.95)	15.25	(0.95)

* Significant at p<.05

As expected, more words were recalled by all groups on the recognition task, compared to the free recall task. This result indicated that the less effortful the task, for all groups, the greater number of words were retrieved.

Tukey's Honestly Significance Difference (HSD) test multiple-comparison procedure was then used for pair-wise comparisons, conditional upon the univariate analysis rejecting the null hypothesis. Individual comparisons with Tukey's (HSD) procedure range at $p<.05$ revealed that, as expected, (a) demented patients differed statistically from the control group on free recall and recognition tasks, (b) demented patients did more poorly than the depressed group on the recognition task, and (c) depressed and control subjects did not differ in their recognition of unrelated words. Results inconsistent with predictions were: (a) demented and depressed subjects showed no difference on the free recall task, and (b) depressed subjects did not perform more poorly than control subjects on the free recall task of unrelated words (see schematic drawing of predicted results Figure 3, Aims and Hypotheses section). The demented group retrieved 10 out of a possible 16 words on the recognition task, unrelated words, which represented a purely chance level result at probability $p<.012$.

Because of the significant sex differences obtained for male subjects on severity of depression, the memory tasks were examined using Pearson Product-Moment Correlation (Table 13). It was found that recognition for unrelated words, only, was significantly poorer for male subjects for combined data across groups ($p = .044$).

In light of research which reports a correlation between severity of depression and severity of memory impairment (Cohen et al., 1982; Henry et al., 1973), the performance on the memory tasks within the depressed group was further examined. Tukey's procedure (range $p<.05$) revealed a significant difference for severity of depressive symptomatology for patients who had not received ECT treatment (Table 14). However, this finding was not reflected on the scores obtained for learning-memory tasks and retrieval conditions.

CHAPTER FOUR – DISCUSSION

The present study was undertaken to examine the nature of memory deficits in dementia and depression, and to test a cognitive model which claimed that the deficits associated with dementia and depression arose from different types of memory failure.

Weingartner et al. (1981) attributed deficits in learning and recall in primary degenerative dementia to an inability to access semantic knowledge. The semantic memory deficit is thought to preclude efficient encoding of new material and thereby limit both acquisition and retrieval. Demented patients would therefore fail to benefit from an inherently structured word list, and demonstrate a failure to recall related words in the form of semantic clusters. By contrast, Weingartner, Cohen, et al. (1981) attributed the memory deficits of depressed subjects to inadequate effort. If new information was structured at the time of learning and recall, depressed patients were expected to perform essentially as control subjects. However, if the task required that the subject initiate effortful encoding and retrieval activities, then the performance of depressed subjects was expected to be worse than controls. Therefore, while demented patients were expected to show acquisition and retrieval problems, depressed subjects were expected to exhibit a more selective pattern of impairment.

It was predicted, based upon Weingartner's hypotheses, that during the initial learning and recall trials, the demented group would score in the impaired range on measures of learning as well as retrieval, whereas the depressed group would exhibit impaired learning and impaired retrieval, but only for tasks requiring sustained effort. The depressed group were also expected to perform essentially as control subjects on the related word list trials, with their performance markedly poorer on the unrelated word list. Further, on delayed recall and recognition testing, it was expected that demented patients would exhibit deficits on all measures, whilst depressed patients would have intact recognition and retention. Substantial enhancement of performance was predicted to result from cueing for depressed patients, but not for individuals with dementia.

4.1 THE ACQUISITION PHASE

Rate of Learning

Demented patients did not show any evidence of learning across trials for either word type. This result was similar to the findings by Weingartner et al. (1981). Learning was evident for the depressed and control subjects. However, the rate at which the learnt differed on the fifth trial of the related word list only, with no initial difference recorded for trial one, or for the rate at which unrelated words were learnt. Thus, the depressed subjects were no poorer at learning on the first trial of both word list types than the control subjects, however they did show a significant difference in the rate of learning on the last trial, relative to the control subjects. Therefore, the final performance of the depressed group could not be predicted from the results on the first trial. This finding is in contrast to Weingartner's results. Why then did the depressed subjects show poorer performance on repeated presentations of the related word trials, compared to normals? Part of the answer may come from the studies by W. R. Miller (1975) and Weingartner et al. (1977) on the effects of lowered motivation and state of arousal on the learning-memory performance for depressed subjects. According to Weingartner et al. (1983), reduction of blood flow in the frontal and temporoparietal association areas may also be held partially responsible for lowered arousal, which in turn, would decrease the attentional capacity of the depressed subject to concentrate sufficiently over the period of five trials. Further, reduction in the noradrenergic system in the depressed patient, which is though to play an important role in the maintenance of arousal levels (Willner, 1985), may also have contributed to the slower rate at which depressed subjects learnt the material, in direct comparison to control subjects. Lowered arousal levels in the depressed subject is also supported when given the knowledge or prior empirical research using L-dopa which was shown to facilitate the performance in verbal learning tasks (Henry et al., 1973: Murphy et al., 1972). The assumption being that even when the task was structured, the depressed subject, because of lowered arousal levels interfering with the attention span, causing vulnerability to distractions and lapses in concentration power, was not able to perform in a comparable manner to that of control subjects. Hart, Kwentus, Taylor, et al. (1987) is one study which gives additional support to the performance of the depressed group, in which it was shown that depressed subjects required longer exposure time to the same material as control subjects to acquire the same information. The fact that these results differ from the Weingartner, Cohen, et al. (1981) study may also be indicative of the younger and less severely depressed group studied by Weingartner, in comparison to the present study, together with the additional known fact of normal decline in cerebral functioning after the age of 60 years in adult subjects (Lezak, 1983).

Recall of related and unrelated words

Demented, together with depressed and healthy elderly subjects, showed greater recall of related words compared to unrelated words. Whilst the depressed and control groups increased word recall across trials, the demented group just failed to obtain a significant learning effect. However, the depressed group did not perform as well as predicted on the related word list, recalling fewer related words than did the control subjects, whereas, they performed better than the predicted outcome on the unrelated word list, relative to controls.

The finding that demented subjects recalled more related than unrelated words is in contrast to the findings of Weingartner et al. (1981). Although the significant effect was small, this result is similar to the findings shown by Cushman and colleagues (1983) of improved recall in dementia patients using semantically related words, in comparison to unrelated word lists. Cushman suggests that this improved recall is due to the remains of some sensitivity to semantic attributes in the mildly demented patient, and that this knowledge is incorporated to facilitate memory performance, albeit to a limited degree. The fact that in the Weingartner study, between 20 and 32 words were presented to his demented group, whilst in the present study a shorter list of 16 words were given, may also serve to explain the difference in findings in the performance of the demented group. This explanation is supported when given the similar findings from the Cushman study which also presented a shorter word list than Weingartner. However, does this suggest that dementia patients actually learnt the semantically organised words better than nonsemantically organised words? To further examine this question, an analysis of the learning-memory trials of demented subjects found that the recall of words from either list type was always the most recently presented items. That is, dementia patients demonstrated a recency effect when recalling words across learning trials, confirming prior research into this area (Martin & Fedio, 1983; Wilson et al., 1983). This suggests that demented patients were functioning on immediate memory only, and therefore, may not have transferred the material into secondary memory. Examination of retrieval cues should help to answer this question. However, this information is of clinical relevance, as it differentiates demented and depressed patients on their organisational strategies for word recall.

The significantly greater number of intrusive items recalled by the demented group, relative to control and depressed subjects confirms prior research (Appel et al., 1982; Fuld et al., 1982) in this field that intrusion errors are not only an important clinical characteristic of these patients' episodic memory disorder, but have also been attributed to an increased sensitivity to proactive interference. That is, to the decremental effect that prior learning has on the retention of subsequently learned material. All intrusive items were semantically similar to the correct item, which explains in part the significant results obtained from the cued retrieval condition.

Results from the present study also support Weingartner's observation that control and depressed subjects would show significantly better recall of the related words, than the unrelated words, and that these two groups would utilise semantic clustering of words during recall over the learning trials. In addition, the results support Weingartner's hypothesis that the depressed group would recall significantly fewer unrelated words in comparison to control subjects. The findings differ, however, from Weingartner's predictions in terms of the performance of the depressed relative to control subjects for both word types in specific areas. The depressed did not perform as well as control subjects in their recall of related words, although the number of words recalled was significantly greater than the number of unrelated words recalled. Rather, this performance pattern is similar to the findings of Levy and Maxwell (1968) who found that depressed subjects did not benefit as much as control subjects from increasing structure. Hertel and Hardin (1990) also present the view that depressed subjects' performance pattern is indicative of a loss of spontaneous use of organisational strategies, which could also explain the different results obtained from this study, relative to the Weingartner, Cohen, et al. (1981) study. The performance of the depressed subjects can also be explained by the arousal-activation state hypothesis as viewed by Henry et al. (1973) and Murphy et al. (1972). This hypothesis is supported by research using the drug d-Amphetamine (Reus et al., 1979), which was found to activate the noradrenergic function and stimulate the level of arousal in depressed patients, thereby prolonging the life of the short-term memory trace, with beneficial effects on word recall.

To test this hypothesis, it may be beneficial for the depressed group to increase the exposure time to each word presented in order to compensate for the effect on the short-term memory trace as suggested by the Reus et al. (1979) and Hart, Kwentus, Taylor, et al. (1987) studies.

Although this lowered state of arousal would have been present in the depressed group studied by Weingartner, research has suggested that severity of depression is correlated with severity of memory impairment (Cohen et al., 1982; Sternberg &b Jarvik, 1976; Stromgren, 1977). It needs to be emphasised that Weingartner's depressed group were moderately depressed, in comparison to the moderately-severely depressed subjects in the present study. Depressed subjects showed significant impairment relative to controls on the recall of unrelated words across trials, a result consistent with research by Weingartner, Cohen et al. (1981), Hasher and Zacks (1979) and Watts et al. (1990). These authors have attributed this result to a reduction in attentional resources, due to a perceived difficulty presented by such an "effortful" type task as an unstructured word list. Hart, Kwentus, Hamer, et al. (1987) provide further support for the results of the present study. They attributed the declined performance on more "effortful" type tasks as being due to a deficit in motivational factors associated with depression. However, the performance by the depressed group on the unrelated word lists was not as poor, in relation to the control group, as had been predicted. This is no doubt due to the comparatively poorer performance achieved by the depressed subjects on the related word list.

The demented group recalled fewer semantic clusters over the five learning trials, relative to depressed and controls, consistent with the views expressed by Jorm (1986b) that attentional resources which require semantic processing are seen to decline in the early stages of the dementia syndrome. This nonsignificant result for the demented subjects is also consistent with findings by other researchers (Bayles, 1982; Butters et al., 1987; Martin & Fedio 1983; La Rue et al., 1986; Ober et al., 1986) with the view that demented patients are not able to demonstrate semantic organisational strategies as efficiently as the other two groups. Semantic clustering reflects the extent to which a subject actively imposes organisation on the related list of words according to shared semantic features. According to Craik (1982), this semantic clustering learning strategy typically results in the most effective encoding into long-term memory. Research by Nebes et al. (1984) also suggests that associational links between semantic concepts is influenced by an intact knowledge of semantic relationships, which in turn must influence the degree to which clustering can be utilised in word recall. It would be evident from the results from this study that the demented subjects failed to cluster semantically related words because of an impairment in these associational links.

The depressed group, as expected, demonstrated significant semantic clustering in their recall of related words across learning trials, as did the control group. Further, both depressed and control subjects utilised clustering of words to facilitate recall of words, increasing the number of clusters with each trial. However, depressed subjects recalled significantly fewer semantic clusters than controls, indicating a lowered level of efficiency in encoding information, even when the material was heavily structured. This performance pattern is similar to that obtained by Levy and Maxwell (1968), who found that depressed subjects demonstrated less benefit from structured material than did control subjects. The discrepancy between this study and the Weingartner, Cohen, et al. (1981) results may also lie with the use of patients of lesser severity in depressive symptomatology, and in the younger aged depressed group used by Weingartner.

4.2 THE RETRIEVAL PHASE

The effect of cues at retrieval

Demented and depressed subjects showed significant improvement in word recall following the presentation of recognition cues, relative to their performance on free recall, whilst the control group demonstrated a similar performance across all retrieval conditions. Cued recall so enhanced the demented subjects' performance that they did not differ significantly from the depressed group, in contrast to Weingartner et al.'s (1981) findings and also to earlier research (Davis & Mumford, 1984; Tuokko & Crockett, 1989) on the nonfacilitating effect of semantic cues on word recall. The findings from the present study are, however, concurrent with the view held by Nebes and Brady (1988) and Nebes et al. (1989) that some semantic knowledge and structure remains in the mildly demented patient. The performance by the demented subjects is also consistent with similar research on semantic cueing by Miller (1975) and Morris et al. (1983), using "structural" cues, to improve memory performance.

It is possible, however, that the dementia patients did not encode the semantic features of the material into secondary memory, as the study by Cushman et al. (1983) would suggest. Rather, the demented subjects may have used the cued recall cue (e.g., clothes) to generate the first word that came to mind, as in a word association task, where the learning is incidental (Nebes et al., 1986; Nebes & Brady, 1988). If demented subjects were operating on immediate memory, rather than encoding material into secondary memory, and given that all intrusive items were of a semantically similar nature to the correct item, the semantic cues presented would further serve to prompt semantic knowledge memory of the demented patient, whereby the search for words fitting the semantic cue would be carried out. That is, the demented subject may merely have thought of the first words which came to mind when given the semantic cue, which would have been the most commonly used words within that category. The fact that semantic cues were helpful to the demented subject in recalling related words, but are not necessarily indicative of having learnt the material, as Tulving and Pearlstone (1966) would suggest, is supported by the view held by Nebes (1989) that incidental learning is unimpaired in dementia patients, and also further explains the nonsignificant effect of semantic clustering demonstrated over the five learning trials by this patient group.

A suggested method to further test the hypothesis that dementia patients may have merely thought of the first word which came to mind when presented aurally with semantic cues, would be to present words of a lower frequency using Kucera and Francis (1967) as a guide. This suggestion is supported by the findings of Barker and Lawson (1968) who found that dementia patients were more impaired on low-frequency words compared to high-frequency words. Research by Davis and Mumford (1984) suggests that cueing the patient with the first letter of each word, rather than providing semantic cues, was more effective. In addition, a longer exposure time to the material may produce enhanced results based upon the work of Becker et al. (1987) and Huppert and Kopelman (1989). Finally, the dementia group may be better served by reducing the number of words to five based upon the research findings of Miller (1971) and Kopelman (1985).

Differences in the results from the present study and Weingartner's study may also reflect on the number of words which comprised each word list. This specific number of words was based on the Rey Auditory Verbal Learning Test (Rey, 1964), and was considered to be easier on the demented patients than a 20 word list which featured in the Weingartner study. In addition, all of the 16 words used in the present study were nouns, comprising no more than six letters in length, and all had a high degree of associability to the category name. If, as Nebes et al. (1986) suggests, some knowledge of semantic associations remains in the early phase of the disorder, high associability of words would serve to increase the likelihood of dementia subjects recalling words without intentionally searching for the correct item.

The demented subjects were worse on the recognition task for both word list types, relative to the depressed and control subjects, as predicted by Weingartner et al. (1981). However, in contrast to the Weingartner study and to prior research that found recognition cues less effective in facilitating memory performance (Morris et al., 1983; Salmon et al., 1989), recognition cues were seen as being more effective than semantic cues for this patient group, since their performance was above chance level. In addition, the demented group showed a significantly improved performance compared to their free recall of words. Difference in performance between the Weingartner study and the present one, may be due to the use of a forced-choice paradigm, based upon the study by Miller (1975), which demonstrated that demented patients were able to benefit from this type of recognition task manipulation. This particular paradigm appears to be effective because it reduces the search the subject is required to make through a number of different alternative answers. That is, a forced-choice recognition paradigm provides a less "effortful" type task, than the recognition of numerous items as in the Salmon et al. (1989) study. It would seem, therefore, that recognition cues are more powerful at facilitating memory performance for the demented subjects than semantic cues, particularly if the number of alternative answers are limited. Further, the results for semantic cues hold some doubt as to whether they in fact facilitated memory of items that had been learnt. The emergence from this study of a significant difference in the recognition of words, both from the related and unrelated word lists, between the demented and depressed groups, highlights a potentially useful task for the differentiation of mild Alzheimer's disease and depression in the elderly patient. No overlap of scores was found between these two groups on this retrieval condition. It is significantly evident, from the scores obtained, that this difference in recognition of words was not because the dementia patients did so poorly. Rather, it was because the depressed group performed so well.

The depressed group performed as well as control subjects on the cued recall and the recognition tasks of both word list types, as consistent with Weingartner's findings, and concurrent with previous literature (Cohen et al., 1982; Hart, Kwentus, Taylor, et al., 1987; La Rue, 1986; Roy-Byrne et al. 1986; O'Connor et al., 1990), using verbal and nonverbal recognition tasks. Further, Hertel and Hardin (1990) found that impairment on recognition tasks was only evident for depressed subjects when the task was not accompanied by detailed instructions on how to proceed. This performance pattern is also consistent with Hasher and Zacks' (1979) conclusion that on less "effortful" tasks, such as recognition, depressed subjects will perform well because of the limited degree of attention required to successfully complete the task. Therefore, the lowered arousal levels which are considered to be experienced by this patient group (Henry et al., 1973; Murphy et al., 1972) do not interfere with the retrieval of processed information. The results are in contrast, however, to the Miller and Lewis (1977) study, which found lowered levels of recognition performance by the depressed subjects, relative to control subjects. Miller and Lewis interpreted their results in the light of the depressed subject's unwillingness to make an error.

The ceiling effect on the recognition task demonstrated by the control group was not unexpected, as previous studies have shown this task to be relatively age immune (Craik, 1977, 1984), due to the supportive nature of environmental cues, and, particularly when given in a forced-choice paradigm, as demonstrated by Miller (1975). The performance of the control subjects was consistent with the predictions made from the Weingartner study, with the framework outlined by Hasher and Zacks (1979) with regard to less "effortful" processing conditions, and to Hertel and Hardin's (1990) study which found that intact initiation of strategies allowed for successful completion of a recognition task.

Delayed Recall

A performance similar to the predictions from the Weingartner et al. (1981) study was found with the demented subjects in relation to control subjects, with significant differences on both word list types. Tukey's contrasts ($p<.05$) show that a greater amount of material was retained by the depressed subjects, compared to demented subjects, for trial five relative to delayed recall on the related word list, but not for unrelated words. That is, the demented and depressed subjects maintained the same forgetting rate of unrelated words between trial five and the delayed recall. This pattern of performance is in contrast to the Weingartner predictions, and may be explained by the research on interference effects. That is, the depressed subjects were disadvantaged by the prior learning of a list of related words. Further, Hart, Kwentus, Taylor, et al. (1987) found that demented subjects, after learning to criterion, showed rapid forgetting in the first 10 minutes, relative to depressed subjects. Thus, although the demented subjects demonstrated recall of more related than unrelated words, their ability to retain this material was less efficient than the depressed group. The work by Hertel and Hardin (1990) may also explain the similarity in delayed recall of unrelated words for the demented and depressed subjects. These authors concluded that deficits seen in the depressed patient arise from a lack of strategies to encode the material. Interference effects may also explain the poorer-than-expected performance for the control subjects on the unrelated words, relative to the depressed subjects. Significant differences between control and depressed subjects, were found on the delayed recall task for related words, consistent with the predictions from Weingartner. In addition, Tukey's contrasts ($p<.05$) found that a significantly greater number of words had been retained by the control subjects, relative to depressed subjects, from trial five to the delayed recall of related words, but not for unrelated words.

4.3 METHODOLOGICAL SECTION

Level of education, in the current study, was similar to that of Weingartner's demented and depressed patients, and therefore, does not account for the contrasting findings in the two studies. Premorbid cognitive functioning, as assessed by the NART (Nelson & O'Connell, 1978), was also similar for both demented and depressed groups within the present study. In fact, the NART was found not to correlate with any variables for any of the groups, in the current study.

Electroconvulsive Therapy (ECT)

The effects of ECT on cognitive functioning is still contentious (e.g., Cronholm & Ottosson, 1963; Squire & Slater, 1983), with some studies reporting positive effects of ECT on memory (e.g., Kendrick & Post, 1967; Stromgren, 1977), whilst other studies report negative effects (e.g., Cronholm & Ottosson, 1963; Squire

& Slater, 1983). Since half the depressed group within the study had been receiving ECT at some time during their hospital stay, this section will examine the research on the effects of ECT on the learning and memory performance of the depressed patient.

Stromgren (1977) found that the harmful effect of unilateral ECT on memory was only very slight. Kendrick and Post (1967) demonstrated that no deleterious effects of ECT were present for depressed patients, when they were required to learn new material 24 hours after treatment. Memory impairment is considered to be greater following bilateral ECT than after right unilateral ECT (Squire & Salter, 1983). However, Fraser and Glass (1980) using a self-rating memory scale, found no difference between unilateral and bilateral ECT, for their depressed group, but did find impaired memory function for the learning of new material prior to ECT. Three weeks post treatment, memory was found to have improved to within normal range for their age. Squire and Zouzounis (1988) reported similar findings in their study of depressed and amnesic patients' perception of their own memory problems.

On the other hand, Cronholm and Ottosson (1961) found that bilateral ECT therapy had an adverse influence mainly on retention of material, whilst depression mainly impaired learning or acquisition of material. However, following ECT therapy (Cronholm & Ottosson, 1963), learning showed an improvement parallel with that of improvement in the depressive symptomatology.

In summary of the above studies on the effect of ECT on learning and memory, it would be apparent that ECT has a slight effect on the learning of new material and on retention of that material, and that this influence appears to be minimal, particularly for unilateral ECT therapy. However, the results would suggest that some caution be exercised in interpreting memory impairments in depressed patients who have received ECT therapy.

In the light of the above knowledge, the learning trials and retrieval tasks of related and unrelated words were subjected to analysis to investigate if any significant differences existed between those patients who had received ECT treatment and those patients who had not. The use of individual Tukey's comparison's (range $p<.05$), revealed no significant differences were evident, within the depressed group on either the related or the unrelated word learning trials and retrieval conditions.

Sample size

The current study may be criticised on the basis of the small sample sizes employed. However, in the majority of published clinical studies on dementia and depression, small numbers of patients have been employed, and often with unequal numbers of subjects. This is a function of the small number of cases available which fulfill established criteria. For example, the Cushman et al, (1988) study used unequal subject numbers with 13 demented patients compared to 17 control subjects. Weingartner, Cohen, et al. (1981) and Weingartner et al. (1981) used 10 depressed and 14 demented patients, respectively, with matched control subjects. The La Rue et al. (1986) study comprised 10 demented and 10 depressed patients, Buschke (1984) had seven patients with Alzheimer's disease, whilst Morris et al. (1983) used 10 demented patients to matched control subjects. Of greater importance than comparing numbers of subjects with other research, is whether the groups' criteria for selection into the study have produced significant effects. Factors, such as age, education level and premorbid cognitive functioning level, are considered to influence performance to a significant degree. There criteria are important in studies dealing with dementia and depression, for researchers to equate all subjects on a base level of performance in order to draw relevant conclusive findings. It has already been noted that none of these factors produced a significant effect on any of the tasks for any of the groups in the present study.

Statistically, using a small sample may create a false impression of the results, however, where appropriate Tukey's procedure was employed to provide a more confident criteria by which to measure the findings. The Kruskal-Wallis was also employed to examine the small number of intrusions and perseverations obtained by the groups. It is acknowledged that utilisation of these statistical measures do not always provide sufficient protection against the probability of a type 1 error.. Larger sample sizes would certainly provide further support for the results obtained in this study.

Floor and ceiling effects in tests

The demented subjects' performance on the related word list may have been obscured by "floor" effects, because their performance was low. If, as Becker et al., (1987) and Huppert and Kopelman (1989) suggest, that acquisition of material is much slower for the demented patient, an increased number of learning trials may have produced a significant learning performance for the categorised word list. This supposition is supported by the fact that the demented subjects just failed to obtain a significant effect for related words.

The ceiling effect obtained by the control subjects on the recognition task may be indicative of the ease with which this group learnt the 16 words, relative to the demented and depressed subjects. However, the primary aim of this study was to compare demented and depressed subjects on the same tasks and therefore a requirement was that these two groups should be given a number of words that was considered to be within a suitable range for them to learn, as specified by Lezak (1983).

4.4 CONCLUSIONS OF STUDY

The findings from this research, therefore, suggest that the use of less "effortful" verbal recall tasks such as cued recall and recognition, facilitate recall of words to a greater extent for dementia patients, than for depressed subjects. The addition of cued recall is generally considered to reveal learning and memory that has otherwise not been demonstrated by free recall (Buschke, 1984). However, the sharp significant increase in word recall with the use of semantic cues found by the demented patients does not necessarily imply that this group have encoded information by its semantic properties. Semantic encoding may be limited and severely impaired, but the categorical relationships between the words may still be apparent to the demented, however, they are unable to encode the words by the use of semantic markers for efficient retrieval, because of an impairment or loss of semantic markers in the lexicon, as suggested by Martin and Fedio (1983) and Schwartz et al. (1979). The failure to store information by semantic markers leads to a decreased ability to recognise and later retrieve that information by the provision of semantic cues which are based on those same semantic categories. The significant increase in word recall by cueing may be simply an artifact effect arising from using high frequency, commonly used words in each category. Suggestions as to alternative methods of isolating this effect have already been discussed. The significantly higher number of intrusive items found for the demented group may also be interpreted as further evidence that categorical cues no longer have the same power to aid in retrieval of associated words.

Therefore, the use of another retrieval condition, namely recognition, provided the opportunity of demonstrating learning by the demented group. Recognition does not require the "effortful" process of a search through long-term memory (Hasher & Zacks, 1979), nor does it rely upon semantic markers to facilitate recall, as in the case of cued recall (Simon, 1976), and is independent of the subject's intention to learn (Kintsch, 1970). Subjects are not required to retrieve items, by carrying out a "search" from long-term memory. Rather, subjects only need compare the item given by the examiner with a vague awareness of having seen a specific item recently. Recognition is the least "effortful" task of the three retrieval conditions used in this study, according to Hasher and Zacks' (1979) framework. With the retrieval demands minimised during the recognition trial, the results of this procedure more accurately reflect the contents of long-term memory, as opposed to being confounded with inability to retrieve the information. The lower performance of demented subjects, compared with depressed and control subjects, on the recognition trial would indicate either that fewer items were making it from immediate memory to secondary memory or that the process of retaining these items in long-term memory was less efficient. Thus, in addition to retrieval difficulty, dementia patients had other encoding or retention problems.

In summary, in the present study, recognition proved to be a better task by which to distinguish the dementia patients' performance, relative to depressed subjects. The demented group performed at an above chance level. Therefore, the deficit lies not just with encoding of material, but also in gaining access to semantic knowledge, which appears to be severely limited. Using a retrieval condition which requires less cognitive capacity, and is therefore, according to Hasher and Zacks (1979), "easier", provided the demented patient with the ability to demonstrate learning. Thus, it may be assumed that for the dementia patient to achieve above chance level of recognition, encoding of material must have occurred. According to Simon (1976), encoding of material into long-term memory requires elaboration of material along semantic lines. If access to semantic knowledge is severely limited, as in the case of demented subjects, it may well be that an encoding problem is not sufficient to explain the deficit suffered by these patients. Included in this explanation must be the fact that some knowledge has been encoded and retrieved by recognition. Therefore, retrieval mechanisms, based upon an inability to access semantic knowledge memory, are important in qualifying the deficit observed in dementia.

A plausible framework for the overall pattern of findings for learning trials of related and unrelated word lists, is that of automatic versus effortful processes (Hasher & Zacks, 1979). In this framework, the recall differences between the groups may reflect the disruption of active learning strategies following dementia and depressive disorders. Thus, while depressive patients were able to appreciate the categorical relationships among words, they were less efficient at using this information to help them remember to the same extent as control subjects. By comparison, the dementia patients, whilst showing benefit from a condition such as a highly structured word list, in their greater word recall for related words, fail to spontaneously apply strategies such as semantic clustering to guide recall. The finding that demented patients neither clustered at recall nor subjectively organised across trials points to their passive approach to learning. Hasher and Zacks' theoretical framework is supported indirectly from research by Craik and Rabinowitz (1984). Their research found that older people had fewer processing resources available, and therefore failed to actively modify novel situations because of the difficulty and effort involved. These findings are consistent with the idea that "effortful" tasks require greater cognitive capacity and as a result, these types of tasks, as opposed to well practised tasks, are the first to show impairment in the aged (Craik, 1984; Hasher & Zacks, 1979; Lewis, 1979).

4.5 CLINICAL IMPLICATIONS OF THIS STUDY

This study is important for three reasons:

(1) both mildly demented and depressed patients hospitalised for their condition, have been compared to healthy elderly control subjects, who have been equated on age, education level, and premorbid cognitive functioning; and

(2) findings from this study show that episodic memory deficits experienced by demented and depressed patients, differ, specifically for recognition task performances for related and unrelated words, for these patient groups.

The implication for clinicians undertaking early diagnosis of mild dementia in the elderly, is that they showed impairment of recognition tasks, in comparison to elderly depressed patients. Not only does recognition represent a differentiating task for elderly mildly demented and depressed patients, but, because of its less "effortful" nature, recognition also presents as a less arduous, less stressful, and less time consuming task, both for the clinician and for the patient.

A prediction that could have been made based upon Weingartner's hypothesis of an encoding deficit for the demented patient, would be that a distinction could have been made on the basis of performances of the demented and depressed patients for the five learning trials of related words. As is apparent in the results of the current study, such a comparison between these two groups, leading to a significant difference, could not have been made, as the demented group did recall a number of related words, although this number did not reach a significant effect.

REFERENCES

Abrams, R., Swartz, C.M., & Vedak, C. (1989). Antidepressant effects of right versus left unilateral ECT and the lateralization theory of ECT action. American Journal of Psychiatry, 146, 1190-1192.

Alexopoulos, G.S. (1989). Editorial. Late-life depression and neurological brain disease. International Journal of Geriatric Psychiatry, 4, 187-190.

Allison, R.S. (1962). The senile brain. London: Edward Arnold.

American Psychiatric Association. (1987). Diagnostic and statistical manual of mental disorders (3rd ed.), Revised. Washington, DC:

Appell, J., Kertesz, A., & Fisman, M. (1982). A study of language in Alzheimer patients. Brain and Language, 17, 73-91.

Baddeley, A.D. (1986). Working memory. London: Oxford University Press.

Baddeley, A.D., & Hitch, G.J. (1974). Working memory. In G. Bower (Ed.), Recent advances in learning and motivation, Vol. VIII. New York: Academic Press.

Baddeley, A.D., Logie, R., Bressi, S., Della Sala, S., & Spinnler, H. (1986). Dementia and the working memory. The Quarterly Journal of Experimental Psychology, 38A, 603-618.

Ball, M.J., Fisman, M., Hachinski, V., Blume, W., Fox, A., Kral, V.A., Kirshen, A.J., & Fox, H. (1985). A new definition of Alzheimer's disease: A hippocampal dementia. The Lancet, 1, 14-16.

Barker, M.G., & Lawson, J.S. (1968). Nominal aphasia in dementia. British Journal of Psychiatry, 114, 1351-1356.

Battig, W.F., & Montague, W.E. (1969). Category norms for verbal items in 56 categories: A replication and extension of the Connecticut category norms. Journal of Experimental Psychology (Monograph, 80(3), 1-46.

Baxter, L.R., Phelps, M.E., Mazziotta, J.C., Schwartz, J.M., Gerner, R.H., Selin, C.E., & Sumida, R.M. (1985). Cerebral metabolic rates for glucose in mood disorders. Archives of General Psychiatry, 42, 441-447.

Bayles, K.A. (1982). Language function in senile dementia. Brain and Language, 16, 265-280.

Bayles, K.A., & Tomoeda, C.K. (1983). Confrontation naming impairment in dementia. Brain and Language, 19, 98-114.

Bayles, K.A., Tomoeda, C.K., & Kasniak, A.W. (1985). Verbal preservation of dementia patients. Brain and Language, 25, 102-116.

Beal, M.R., Mazurek, M.F., Tran, V.T., Chattha, G., Bird, E.D., & Martin, J.G. (1985). Reduced numbers of somatostatin receptors in the cerebral cortex in Alzheimer's disease. Science, 229, 289-291.

Becker, J.T. (1988). Working memory and secondary memory deficits in Alzheimer's disease. Journal of Clinical and Experimental Neuropsychology, 10(6), 739-753.

Becker, J.T., Boller, F., Saxton, J., & McGonigle-Gibson, K.L. (1987). Normal rates of forgetting of verbal and non-verbal material in Alzheimer's disease. Cortex, 23, 59-72.

Blessed, G., Tomlinson, B.E., & Roth, A. (1968). The association between quantitative measures of dementia and of senile change in the cerebral grey matter of elderly subjects. British Journal of Psychiatry, 114, 797-911.

Bowen, D.M., Smith, C.B., White, P., Davison, A.N. (1976). Neurotransmitter-related enzymes and indices of hypoxia in senile dementia and other abiotrophies. Brain, 99, 459-496.

Bowles, N.L., Obler, L.K., & Albert, M.L. (1987). Naming errors in healthy aging and dementia of the Alzheimer type. Cortex, 23, 519-524.

Breitner, J.C.S., & Folstein, M.F. (1984). Familial Alzheimer dementia: A prevalent disorder with specific clinical features. Psychological Medicine, 14, 63-80.

Brink, T.A., Yesavage, J.A., Lum, O., Heersema, P., Adey, M., & Rose, T.L. (1982). Screening tests for geriatric depression. Clinical Gerontologist, 1, 37-44.

Burvill, P.W. (1988). Psychiatric assessment of the elderly. In R.W. Warne and D.M. Prinsley (Eds.), A manual of geriatric care (pp. 53-65). Sydney: Williams and Wilkins & Associates.

Burvill, P.W., Stampfer, H., & Hall, W. (1986). Does depressive illness in the elderly have a poor prognosis? A review. Australian and New Zealand Journal of Psychiatry, 20, 422-427.

Buchsbaum, M.S., DeLisi, L.E., Holcomb, H.H., Cappelletti, J., Johnson, J., King, A.C., Hazlett, E., Dowling-Zimmerman, S., Post, R.M., Morihisa, J., Carpenter, W., Cohen, R., Pickar, D., Weinberger, D.R., Margonlin, R., & Kessler, R.M. (1984). Anteroposterior gradients in cerebral glucose use in schizophrenia and affective disorders. Archives of General Psychiatry, 41, 1159-1166.

Buschke, H. (1973). Selective reminding for analysis of memory and learning. Journal of Verbal Learning and Verbal Behavior, 12, 543-550.

Buschke, H. (1984). Cued recall in amnesia. Journal of Clinical Neuropsychology, 6, 433-436.

Butters, N., Granholm, E., Salmon, D.P., Grant, I., & Wolfe, J. (1984). Episodic and semantic memory: A comparison of amnesia and demented patients. Journal of Clinical and Experimental Neuropsychology, 9(5), 479-497.

Caine, E.D., Weingartner, H., Ludlow, C.L., Cudahy, E.A., & Wehry, S. (1981). Qualitative analysis of scopolamine-induced amnesia. Psychopharmacology, 74, 74-80.

Caramazza, A., & Berndt, R.S. (1978). Semantic and syntactic processes in aphasia: A review of the literature. Psychological Bulletin, 85, 898-918.

Cawley, R.H., Post, F., & Whitehead, A. (1973). Barbiturate tolerance and psychosocial functioning in elderly depressed patients. Psychological Medicine, 3, 39-52.

Cermak, L.S. (1979). Amnesic patient's level of processing. In L.S. Cermak, & F.I.M. Craik (Eds.), Levels of processing in human learning. Hillsdale, N.J.: Erlbaum.

Cermak, L.S. (1984). The episodic-semantic distinction in amnesia. In L.R. Squire & N. Butters (Eds.), Neuropsychology of memory. New York: Guilford Press.

Cohen, N.J., & Squire, L.R. (1980). Preserved learning and retention of pattern-analyzing skill in amnesia: Dissociation of knowing how and knowing that. Science, 210, 207-210.

Cohen, N.J., Weingartner, H., Smallberg, S.A., Pickar, D., & Murphy, D.L. (1982). Effort and cognition in depression. Archives of General Psychiatry, 39, 593-597.

Cole, K.D., & Zarit, S.H. (1984). Psychological deficits in depressed medical patients. The Journal of Nervous and Mental Disease, 172(3), 150-155.

Corkin, S. (1982). Some relationships between global amnesias and the memory impairment in Alzheimer's disease. In S. Corkin, K.L. Davis, J.H. Growden, E. Usdin, & R.J. Wurtman (Eds.), Alzheimer's disease: A report of research in progress (pp. 149-154). New York: Raven Press.

Corkin, S., Gabrieli, J.D.E., Stanger, B.Z., Mickel, S.F., Rosen, J., & Sullivan, E.V. (1986). Skill learning and priming in Alzheimer's disease (AD). Neurology, 36 (Suppl. 1.), 296.

Corsellis, J.A.N. (1970). The limbic areas in Alzheimer's disease and in other conditions associated with dementia. The G.E.W. Wolstenholm & M. O'Connor (Eds.), Alzheimer's disease and related conditions. London: J.&A. Churchill.

Corsellis, J.A.N. (1976). Ageing and the dementias. In W. Blackwood & J.A.N. Corsellis (Eds.), Greenfield's neuropathology. Chicago: Year Book Medical Publishers.

Coughlan, A.K., & Hollows, S.E. (1984). Use of memory tests in differentiating organic disorder from depression. British Journal of Psychiatry, 145, 164-167.

Craik, F.I.M. (1977). Age differences in human memory. In J.E. Birren & K.W. Schaie (Eds.), Handbook of the psychology of aging. New York: Van Nostrand Reinhold.

Craik, F.I.M. (1984). Age differences in remembering. In L.R. Squire & N. Butters (Eds.), Neuropsychology of memory. New York: Guilford Press.

Craik, F.I.M., & Lockhart, R.S. (1972). Levels of processing: A framework for memory research. Journal of Verbal Learning and Verbal Behavior, 11, 671-684.

Craik, F.I.M., & Rabinowitz, J.C. (1984). Age differences in the acquisition and use of verbal information. In H. Bouma & D.G. Bouwhuis (Eds.), Attention and performance X. Hillsdale, N.J.: Erlbaum.

Craik, F.I.M., & Tulving, E. (1975). Depth of processing and the retention of words in episodic memory. Journal of Experimental Psychology: General, 104(3), 268-294.

Cronholm, B., & Ottosson, J-O. (1961). Memory functions in endogenous depression: Before and after electroconvulsive therapy. Archives of General Psychiatry, 5, 193-199.

Cronholm, B., & Ottosson, J-O. (1963). The experience of memory function after electroconvulsive therapy. British Journal of Psychiatry, 190, 251-258.

Cummings, J.L., & Benson, D.F. (1983). Dementia: A clinical approach. Boston: Butterworths.

Cummings, J.L., & Benson, D.F. (1988). Psychological dysfunction accompanying subcortical dementia. Annual Review of Medicine, 39, 53-61.

Cushman, L.A., Como, P.G., Booth, J., & Caine, E.D. (1988). Cued recall and release from proactive interference in Alzheimer's disease. Journal of Clinical and Experimental Neuropsychology, 10(6), 685-692.

Davies, P.E., & Maloney, A.J. (1976). Selective loss of central cholinergic neurons in Alzheimer's disease. The Lancet, ii, 1403.

Davis, P.E., & Mumford, S.J. (1984). Cued recall and the nature of the memory disorder in dementia. British Journal of Psychiatry, 144, 383-386.

D'Elia, G., & Perris, C. (1973). Cerebral function dominance and depression. Acta Psychiatrica Scandinavia, 49, 191-197.

Delis, D.C., Kramer, J.H., Kaplan, E., & Ober, B.A. (1987). California verbal learning test manual. New York: The Psychological Corporation.

Drachman, D.A., & Leavitt, J. (1974). Human memory and the cholinergic system: A relationship to aging? Archives of Neurology, 30, 113.

Eaton, W.W., Regier, D.A., Locke, B.Z., & Taube, C.A. (1981). The epidemiological catchment area program of the national institute of mental health. Public Health Reports, 96, 319-322.

Eslinger, P.J., & Damasio, A.R. (1986). Preserved motor learning in Alzheimer's disease: Implications for anatomy and behavior. Journal of Neuroscience, 6(10), 3006-3009.

Fillenbaum, G.G., Hughes, D.C., Heyman, A., George, L.K., & Blazer, D.G. (1988). Relationships of health and demographic characteristics to mini-mental state examination score among community residents. Psychological Medicine, 18, 719-726.

Filley, C.M., Kelly, J., & Heaton, R.K. (1986). Neuropsychologic features of early-and-late-onset Alzheimer's disease. Archives of Neurology, 43, 574-576.

Folstein, M.F., Folstein, S.E., & McHugh, P.R. (1975). "Mini-mental state": A practical method for grading the cognitive state of patients for the clinician. Journal of Psychiatric Research, 12, 189-198.

Foulds, G.A. (1952). Temperamental differences in maze performance: Part II. The effect of distraction and electroconvulsive therapy on psychomotor retardation. British Journal of Psychology, 43, 33-41.

Fraser, R.M., & Glass, I.B. (1980). Unilateral and bilateral ECT in elderly patients. Acta Psychiatrica Scandinavica, 62, 13-21.

Fuld, P.A., Katzman, R., Davies, P., & Terry, R.O. (1982). Intrusions as a sign of Alzheimer dementia: Clinical and pathological verification. Annals of Neurology, 11, 155-159.

Gibson, A.J. (1981), A further analysis of memory loss in dementia and depression in the elderly. British Journal of Clinical Psychology, 20, 179-185.

Gibson, A.J., & Kendrick, D.C. (1976). The development of a visual learning test to replace the SLT in the Kendrick Battery. Bulletin of the British Psychological Society, 29, 200-201.

Graf, P., & Schacter, D.L. (1985). Implicit and explicit memory for new associations in normal and amnesic subjects. Journal of Experimental Psychology: Learning, Memory and Cognition, 11(3), 501-518.

Graf, P., Shimamura, A.P., & Squire, L.R. (1985). Priming across modalities and priming across category levels: Extending the domain of preserved function in amnesia. Journal of Experimental Psychology: Learning, Memory and Cognition, 11(3), 501-518.

Greene, R.L. (1986). Word stems as cues in recall and completion tasks. The Quarterly Journal of Experimental Psychology, Section A: Human Experimental Psychology, 38A(4), 663-672.

Greenwald, B.S., Kramer-Ginsberg, E., Marin, D.B., Laitman, L.B., Hermann, C.K., Mohs, R.C., & Davis, K.L. (1989). Dementia with coexistent major depression. American Journal of Psychiatry, 146, 1472-1478.

Grimes, D. (1988). The funding of services for the elderly Australian-Influence of health care systems, past anomalies and future prospects. In R.W. Warne & D.M. Prinsley (Eds.), A manual of geriatric care (pp. 510-516). Sydney: William and Wilkins & Associates.

Grober, E., & Buschke, H. (1987). Genuine memory deficits in dementia. Developmental Neuropsychology, 3(1), 13-36.

Gur, R.E., Skolnick, B.E., Gur, R.C., Caroff, S., Rieger, W., Obrist, W.D., Younkin, D., Reivich, M. (1984). Brain function in psychiatric disorders, II: Regional cerebral blood flow in medicated unipolar depressives. Archives of General Psychiatry, 41, 695-699.

Gustafson, L., Hagberg, B., & Ingvar, D.H. (1978). Speech disturbances in presenile dementia related to local cerebral blood flow abnormalities in the dominant hemisphere. Brain and Language, 5, 103-118.

Hachinski, V.C., Lassen, N.A., & Marshall, J. (1974). Multi-infarct dementia. A cause of mental deterioration in the elderly. The Lancet, 2, 207-210.

Hart, R.P., Kwentus, J.A., Hamer, R.M., & Taylor, J.R. (1987). Selective reminding procedure in dementia and depression. Journal of Consulting and Clinical Psychology, 5(1), 111-115.

Hart, R.P., Kwentus, J.A., Taylor, J.R., & Harkins, S.W. (1987). Rate of forgetting in dementia and depression. Journal of Consulting and Clinical Psychology, 5(1), 101-105.

Hasher, L., & Zacks, R.T. (1979). Automatic and effortful processes in memory. Journal of Experimental Psychology: General, 108(3), 356-388.

Henderson, A. (1983). The coming epidemic of dementia. Australian and New Zealand Journal of Psychiatry, 17, 117-127.

Henderson, A., & Jorm, A.F. (1986). The problem of dementia in Australia. Canberra: Australian Government Publishing Service.

Henry, G.M., Weingartner, H., & Murphy, D.L. (1973). Influence of affective states and psychoactive drugs on verbal learning and memory. American Journal of Psychiatry, 130(9), 966-971.

Hertel, P.T., & Hardin, T.S. (1990). Remembering with and without awarenes in a depressed mood: Evidence of deficits in initiative. Journal of Experimental Psychology: General, 119(1), 45-59.

Heston, L.L. (1984). Down's syndrome and Alzheimer's dementia: Defining an association. Psychiatric Developments, 4, 287-294.

Heston, L.L., & White, J.A. (1983). Dementia. A practical guide to Alzheimer's disease and related illness. New York: W.H. Freeman & Company.

Hirschfeld, R.M., Klerman, G.L., Lavori, P., Keller, M.B., Griffith, P., & Coryell, W. (1989). Premorbid personality assessments of first onset of major depression. Archives of General Psychiatry, 46, 345-350.

Huppert, F.A., & Kopelman, M.D. (1989). Rates of forgetting in normal ageing: A comparison with dementia. Neuropsychologica, 27(6), 849-860.

Huppert, F.A., & Tym, E. (1986). Clinical and neuropsychological assessment of dementia. British Medical Bulletin, 42, 11-18.

Ineichen, B. (1987). Measuring the rising tide. How many dementia cases will there be by 2001? British Journal of Psychiatry, 150, 193-200.

Jacoby, L.L. Baker, J.G., & Books, L.R. (1989). Episodic effects on picture identification: Implications for theories of concept learning and theories of memory. Journal of Experimental Psychology: Learning, Memory and Cognition, 15(2), 275-281.

Jacoby, L.L., & Witherspoon, D. (1982). Remembering without awareness. Canadian Journal of Psychology, 36(2), 300-324.

Jorm, A.F. (1986a). Cognitive deficit in the depressed elderly: A review of some basic unresolved issues. Australian and New Zealand Journal of Psychiatry, 20, 11-22.

Jorm, A.F. (1986b). Controlled and automatic information processing in senile dementia: A review. Psychological Medicine, 16, 77-88.

Jorm, A.F. (1987). Understanding senile dementia. Sydney: Croom Helm.

Jorm, A.F., & Korten, A.E. (1988). A method of calculating projected increases in the number of dementia sufferers. Australian and New Zealand Journal of Psychiatry, 22, 183-189.

Jorm, A.F., Korten, A.E., & Henderson, A.S. (1987). The prevalence of dementia: A quantitative integration of the literature. Acta Psychiatrica Scandinavica, 76, 465-479.

Jorm, A.F., Scott, R., Henderson, A.S., & Kay, D.W.K. (1988). Educational level differences on the mini-mental state: The role of test bias. Psychological Medicine, 18, 727-731.

Kang, J., Lemaire, H-G., Unterback, A., Salbaum, J.M., Master, C.L., Grzeschik, K-H, Multhaup, G., Beyeuther, K., & Muller-Hill, B. (1987). The precursor of Alzheimer's disease amyloid A4 protein resembles a cell-surface receptor. Nature, 325, 733-736.

Kay, D.W.K. (1989). Ageing of the population: Measuring the need for care. Age and Ageing, 18, 73-76.

Kempler, W., Curtiss, S., & Jackson, C. (1987). Syntactic preservation in Alzheimer's disease. Journal of Speech and Hearing, 30 (Sept.), 345-350.

Kendrick, D.C., & Post, F. (1967). Differences in cognitive status between health, psychiatrically ill, and diffusely brain-damaged elderly subjects. British Journal of Psychiatry, 113, 75-81.

Keppel, G. (1982). Design and analysis. A researcher's handbook. New Jersey: Prentice-Hall.

Kintsch, W. (1970). Models for free recall and recognition. In D.A. Norman (Ed.), Models of human memory. New York: Academic Press.

Knesevich, J.W., Roro, F.R., Morris, J.C., & LaBarge, E. (1985). Aphasia, family history, and the longitudinal course of senile dementia of the Alzheimer type. Psychiatry Research, 14(3), 255-263.

Knopman, D.S., & Nissen, M.J. (1987). Implicit learning in patients with probable Alzheimer's disease. Neurology, 37, 784-788.

Kopelman, M.D. (1985). Multiple memory deficits in Alzheimer-type dementia: Implications for pharmacotherapy. Psychological Medicine, 15, 527-541.

Kopelman, M.D. (1986). Clinical tests of memory. British Journal of Psychiatry, 148, 517-525.

Kucera, H., & Francis, W.N. (1967). Computational analysis of present-day American English. Providence, Rhode Island: Brown University Press.

LaRue, A. (1989). Patterns of performance on the Fuld object memory evaluation in elderly inpatients with depression or dementia. Journal of Clinical and Experimental Neuropsychology, 11(4), 409-422.

LaRue, A., D'Elia, L.F., Clark, E.O., Spar, J.E., & Jarvik, L.F. (1986). Clinical tests of memory in dementia, depression and healthy aging. Journal of Psychology and Aging, 1(1), 69-77.

Lazarus, L.W., Newton, N., Cohler, B., Lesser, J., & Schweon, C. (1987). Frequency and presentation of depressive symptoms in patients with primary degenerative dementia. American Journal of Psychiatry, 144, 41-45.

Levy, R., & Maxwell, A.E. (1968). The effect of verbal context on the recall of Schizophrenics and other psychiatric patients. British Journal of Psychiatry, 114, 311-316.

Lewis, D.J. (1979). Psychobiology of active and inactive memory. Psychological Bulletin, 86(5), 1054-1083.

Lezak, M.D. (1983). Neuropsychological assessment. New York: University Press.

Lishman, W.A. (1978). Organic psychiatry. London: Blackwell Scientific Publications.

Liston, E.H. (1977). Occult presenile dementia. The Journal of Nervous and Mental Disease, 164(4), 263-267.

Liston, E.H. (1979). Clinical findings in presenile dementia: A report of 50 cases. Journal of Nervous and Mental Disorders, 167, 337-342.

Mahendra, B. (1984). Dementia. A survey of the syndrome of dementia. Lancaster: MPT Press.

Mann, D.M.A., & Yates, P.O., (1986). Neurotransmitter deficits in Alzheimer's disease and in other dementing disorders. Human Neurobiology, 5, 147-158.

Marsden, C.D., & Harrison, M.J.G. (1972). Outcome of investigation of patients with presenile dementia. British Medical Journal, 2, 249-252.

Martin, A., & Fedio, P. (1983). Word production and comprehension in Alzehimer's disease: The breakdown of semantic knowledge. Brain and Language, 19, 124-141.

McAllister, T.W., & Price, T.R.P. (1982). Severe depressive pseudodementia with and without dementia. American Journal of Psychiatry, 139(5), 626-629.

McLean, S. (1987). Review. Assessing dementia. Part I: Difficulties, definitions and differential diagnosis. Australian and New Zealand Journal of Psychiatry, 21, 142-172.

Miller, E. (1971). On the nature of the memory disorder in presenile dementia. Neuropsychologia, 9, 75-78.

Miller, E. (1975). Impaired recall and the memory disturbance in presenile dementia. British Journal of Social and Clinical Psychology, 14, 73-79.

Miller, E. (1981). The nature of the cognitive deficit in senile dementia. Aging, 15, 103-199.

Miller, E., & Lewis, P. (1977). Recognition memory in elderly patients with depression and dementia: A signal detection analysis. Journal of Abnormal Psychology, 86(1), 84-86.

Miller, W.R. (1975). Psychological deficit in depression. Psychological Bulletin, 82(2), 238-260.

Mitchell, D.B. (1989). How many memory systems? Evidence from aging. Journal of Experimental Psychology: Learning, Memory and Cognition, 15(1), 31-49.

Mitchell, D.B., & Perlmutter, M. (1986). Semantic activation and episodic memory: Age similarities and differences. Developmental Psychology, 22(1). 86-94.

Morris, R. (1986). Short-term forgetting in senile dementia of the Alzheimer's type. Cognitive Neuropsychology, 3, 77-97.

Morris, R., Wheatley, J., & Britton, P. (1983). Retrieval from long-term memory in senile dementia: Cued recall revisited. British Journal of Clinical Psychology, 22, 141-142.

Morris, R.G., & Bladdeley, A.D., (1988). Primary and working-memory functioning in Alzheimer-type dementia. Journal of Clinical and Experimential Psychology, 10(2), 279-296.

Morris, R.G., & Kopelman, M.D. (1986). The memory deficits in Alzheimer-type dementia: A review. The Quarterly Journal of Experimental Psychology, 38A, 575-602.

Moscovitch, M. (1984). The sufficient conditions for demonstrating preserved memory in amnesia: A task analysis. In L.R. Squire & N. Butters (Eds.), Neuropsychology of memory. New York: Guilford.

Moscovitch, M., Wiinocur, G., & McLachlan, D. (1986). Memory as assessed by recognition and reading time in normal and memory-impaired people with Alzheimer's disease and other neurological disorders. Journal of Experimental Psychology: General, 115,(4), 331-347.

Murphy, D.L., Henry, G.M., & Weingartner, H. (1972). Catecholamines and memory: Enhanced verbal learning during L-DOPA administration. Psychopharmacologica (Berlin), 27, 319-326.

Murphy, E. (1982). Social origins of depression on old age. British Journal of Psychiatry, 141, 135-142.

Murphy, E. (1983). The prognosis of depression in old age. British Journal of Psychiatry, 142, 111-119.

Nebes, R.D., Boller, F., & Holland, A. (1986). Use of semantic context by patients with Alzheimer's disease. Psychology and Aging, 1, 261-269.

Nebes, R.D., & Brady, C.B. (1988). Integrity of semantic fields in Alzheimer's disease. Cortex, 24, 291-299.

Nebes, R.D., Brady, C.B., & Huff, F.J. (1989). Automatic and attentional mechanisms of semantic priming in Alzheimer's disease. Journal of Clinical and Experimental Neuropsychology, 11(2), 219-230.

Nebes, R.D., Martin, D.C., & Horn, L.C. (1984). Sparing of semantic memory in Alzheimer's disease. Journal of Abnormal Psychology, 93, 321-330.

Nelson, H.E., & McKenna, P. (1975). The use of current reading ability in the assessment of dementia. British Journal of Social Clinical Psychology, 14, 259-267.

Nelson, H.E., & O'Connell, A. (1978). Dementia: The estimation of premorbid intelligence levels using the New Adult Reading Test. Cortex, 14, 234-244.

Nott, P.N., & Fleminger, J.J. (1975). Presenile dementia: The difficulties of early diagnosis. Acta Psychiatrica Scandinavica, 51, 210-217.

Ober, B.A., Dronkers, N.F., Koss, E., Delis, D.C., & Friedland, R.P. (1986). Retrieval from semantic memory in Alzheimer-type dementia. Journal of Clinical and Experimental Neuropsychology, 8, 75-92.

O'Carroll, R.E. (1987). The inter-rater reliability of the National Adult Reading Test (NART): A pilot study. British Journal of Clinical Psychology, 26, 229-230.

O'Connor, D.W., Pollitt, P.A., Roth, M., Brook, P.B., & Reiss, B.B. (1990). Memory complaints and impairment in normal, depressed, and demented elderly persons identified in a community survey. Archives of General Psychiatry, 47, 224-227.

O'Hara, M.W., Hinrichs, J.V., Kohout, K.J., Wallace, R.B., & Lemke, J.H. (1986). Memory complaint and memory performance in the depressed elderly. Psychology and Aging, 1(3), 208-214.

O'Keefe, J., & Nadel, L. (1978). The hippocampus as a cognitive map. London: Oxford University Press.

Ojeda, V.J., Mastaglia, F.L., & Kakulas, B.A. (1986). Causes of organic dementia: A necropsy survey of 60 cases. The Medical Journal of Australia, 145, 69-71.

Partridge, F.M., Knight, R.G., & Feehan, M. (1990). Direct and indirect memory performance in patients with senile dementia. Psychological Medicine, 20, 111-118.

Perlmutter, M., & Mitchell, D.B. (1982). The appearance and disappearance of age differences in adult memory. In F.I.M. Craik & S. Trehub (Eds.), Aging and cognitive processes (pp.127-144). New York: Plenum.

Perris, C. (1975). EEG techniques in the measurement of the severity of depressive syndromes. Neuropsychobiology, 1, 16-25.

Perry, E.K., Perry, R.H., Blessed, G., & Tomlinson, B.E. (1977). Necropsy evidence of central cholinergic deficits in senile dementia. The Lancet, 1, 189.

Popkin, S.J., Gallagher, D., Thompson, L.W., & Moore, M. (1982). Memory complaint and performance in normal and depressed older adults. Experimental Aging Research, 8(30), 141-145.

Reifler, B.V. (1986). Mixed cognitive-affective disturbances in the elderly: A new classification. Journal of Clinical Psychiatry, 47, 354-356.

Reifler, B.V. (1988). Clinical problems in geriatric psychiatry. The relationship between dementia of the Alzheimer type and depression. North Carolina Medical Journal, 49(10), 536-538.

Reifler, B.V., Larson, E., & Hanley, R. (1982). Coexistence of cognitive impairment and depression in geriatric outpatients. American Journal of Psychiatry, 139(5), 623-626.

Reus, V.I., Silberman, E., Post, R.M., & Weingartner, H. (1979). d-Amphetamine: Effects on memory in a depressed population. Biological Psychiatry, 14(2), 345-356.

Rey, A. (1964). L'examen clinique en psychologie (The clinical psychology examination). Paris: Presses Universitaries de France.

Reikkinen, P.J., Laulumaa, V., Sirvio, J., Soininen, H., & Helkala, E.L. (1987). Recent progress in the research of Alzheimer's disease. Medical Biology, 65, 83-88.

Ron, M.A., Toone, B.K., Garralda, M.E., & Lishman, W.A. (1979). Diagnostic accuracy in presenile dementia. British Journal of Psychiatry, 134, 161-168.

Rosenhan, D.L., & Seligman, M.E.P. (1984). Abnormal psychology. New York: W.W. Norten & Company.

Rossor, M.N. (1982). Neurotransmitters and CNS disease. The Lancet, 27, 1200-1204.

Roth, M. (1986). The association of clinical and neurological findings and its bearing on the classification and aetiology of Alzheimer's disease. British Medical Bulletin, 42, 42-50.

Roth, M., Tym, E., Mountjoy, C.O., Huppert, F.A., Hendri, H., Verma, S., & Goddard, R. (1986). Camdex: A standardised instrument for the diagnosis of mental disorder in the elderly with special reference to the early detection of dementia. British Journal of Psychiatry, 149, 698-709.

Rovner, B.W., Broadhead, J., Spencer, M., Carson, K., & Folstein, M.F. (1989). Depression and Alzheimer's disease. American Journal of Psychiatry, 146, 350-353.

Roy-Byrne, P.P., Weingartner, H., Bierer, L.M., Thompson, K., & Post, R.M. (1986). Effortful and automatic cognitive processes in depression. Archives of General Psychiatry, 43, 265-267.

Royal College of Physicians of London, Committee on Geriatrics. (1981). Organic mental impairment in the elderly. Implications for research, education and the provision of services. Journal of the Royal College of Physicians of London, 15, 141-167.

Sackheim, H.A., Prohovnik, I., Moeller, J.R., Brown, R.P., Apter, S., Prudie, J., Devanannd, D.P., & Mukherjee, S. (1990). Regional cerebral blood flow in mood disorders. Archives of General Psychiatry, 47, 60-70.

Salmon, D.P., Granholm, E., McCullough, D., Butters, N., & Grant, I. (1989). Recognition memory span in mildly and moderately demented patients with Alzheimer's disease. Journal of Clinical and Experimental Neuropsychology, 11(4), 429-443.

Salmon, D.P., Shimamura, A.P., Butters, N., & Smith, S. (1988). Lexical and semantic priming deficits in patients with Alzheimer's disease. Journal of Clinical and Experimental Neuropsychology, 10(4), 477-494.

Schacter, D.L. (1985). Priming of old and new knowledge in amnesic patients and normal subjects. Annals of the New York Academy of Sciences, 444, 41-53.

Schacter, D.L., & Graf, P. (1986a). Effects of elaborative processing on implicit and explicit memory for new associations. Journal of Experimental Psychology: Learning, Memory and Cognition, 12(3), 432-444.

Schacter, D.L., & Graf, P. (1986b). Preserved learning in amnesic patients: Perspectives from research on direct priming. Journal of Clinical and Experimental Neuropsychology, 8(1), 727-743.

Schwartz, M.F., Marin, O.S.M., & Saffran, E.N. (1979). Disassociation of language function in dementia: A case study. Brain and Language, 7, 277-306.

Seligman, M.E.P., Klein, D.C., & Miller, W.R. (1975). Depression. In H. Leitenberg (Ed.), Handbook of behavior therapy. New York: Appleton-Century-Crofts.

Shiffrin, R.M., & Schneider, W. (1977). Controlled and automatic human information processing. II. Perceptual learning, automatic attending and a general theory. Psychological Review, 84, 127-190.

Shimamura, A.P. (1986). Priming effects in amnesia: Evidence for a dissociable memory function. The Quarterly Journal of Experimental Psychology, 38A, 619-644.

Shimamura, A.P., Salmon, D.P., Squire, L.R., & Butters, N. (1987). Memory dysfunction and word priming in dementia and amnesia. Behavioral Neuroscience, 101(3), 347-351.

Shimamura, A.P., & Squire, L.R. (1984). Paired-associate learning and priming effects in amnesia: A neuropsychological study. Journal of Experimental Psychology: General, 113(4), 556-570.

Silberman, E.K., Weingartner, H., & Post, R.M. (1983). Thinking disorder in an abstract reasoning task. Archives of General Psychiatry, 40, 775-780.

Silverskiold, P., & Risberg, J. (1989). Regional cerebral blood flow in depression and mania. Archives of General Psychiatry, 46, 253-259.

Sim, M., & Sussman, I. (1962). Alzheimer's disease: Its natural history and differential diagnosis. Journal of Nervous and Mental Disorders, 135, 489-499.

Simon, H.A. (1976). The information-storage system called "human memory". In M.E. Rosenzweig & E.C. Bennett (Eds.), Neural mechanisms of learning and memory (pp. 79-96). Massachusetts: Massachusetts Institute of Technology Press.

Sitaram, N., Weingartner, H., & Gillin, J.C. (1978). Human serial learning: Enhancement with arecoline and choline and impairment with scopolamine. Science, 201, 274-276.

Smith, J.S., & Kiloh, L.G. (1981). The investigation of dementia: Results in 200 consecutive admissions. The Lancet, 2, 824-827.

Snodgrass, J.G., & Corwin, J. (1988). Pragmatics of measuring recognition memory: Applications to dementia and amnesia. Journal of Experimental Psychology: General, 117(1), 34-50.

SPSS-X. (1988). Statistical packages for the social sciences – user's guide. New York: McGraw-Hill Book Company.

Squire, L.R., Shimamura, A.P., & Graf, P. (1985). Independence of recognition memory and priming effects: A neuropsychological analysis. Journal of Experimental Psychology: Learning, Memory and Cognition, 11(1), 37-44.

Squire, L.R., & Slater, P.C. (1983). Electroconvulsive therapy and complaints of memory dysfunction: A prospective three-year follow-up study. British Journal of Psychiatry, 142, 1-8.

Squire, L.R., & Zouzounis, J.A. (1988). Self-ratings of memory dysfunction: Different findings in depression and amnesia. Journal of Clinical and Experimental Neuropsychology, 10(6), 727-738.

Sternberg, D.E., & Jarvik, M.E. (1976). Memory functions in depression. Improvement with antidepressant medication. Archives of General Psychiatry, 33, 219-224.

Stromberg, L.S. (1977). The influence of depression on memory. Acta Psychiatrica Scandinavica, 56, 109-128.

Teng, E.L., Chui, H.C., Schneider, L.S., & Metzger, L.E. (1987). Alzheimer's dementia: Performance on the mini-mental state examination. Journal of the American Geriatric Society, 36, 1-6.

Tomlinson, B.E., Blessed, G., & Roth, M. (1970). Observations on the brains of demented old people. Journal of Neurological Science, 11, 205-242.

Tulving, E. (1972). Episodic and semantic memory. In E. Tulving & W. Donaldson (Eds.), Organisation and memory (pp. 381-403). New York: Academic Press.

Tulving, E. (1985). How many memory systems are there? American Psychologist, 40, 385-398.

Tulving, E., & Pearlstone, Z. (1966). Availability versus accessibility of information in memory for words. Journal of Verbal Learning and Verbal Behavior, 5, 67-80.

Tulving, E., Schacter, D.L., & Stark, H.A. (1982). Priming effects in world-fragment completion are independent of recognition memory. Journal of Clinical and Experimental Psychology, 8(4), 336-342.

Tulving, E., & Thomson, D.M. (1973). Encoding specificity and retrieval processes in episodic memory. Psychological Review, 80(5), 336-342.

Tuokko, H., & Crockett, D. (1989). Cued recall and memory disorders in dementia. Journal of Clinical and Experimental Neuropsychology, 11(2), 278-294.

Tyrrell, P., & Rossor, M. (1988). Recent advances in the understanding of dementia. Postgraduate Medical Journal, 64, 99-106.

Watts, F.N., Dalgleish, T., Bourke, P., & Healy, D. (1990). Memory deficit in clinical depression: Processing resources and the structure of materials. Psychological Medicine, 20, 345-349.

Wechsler, D. (1981). Wechsler adult intelligence scale-revised. New York: The Psychological Corporation.

Weingartner, H. (1984). Psychobiological determinants of memory failures. In L.R. Squire & N. Butters (Eds.), Neuropsychology of memory. New York: Guilford Press.

Weingartner, H. (1985). Models of memory dysfunctions. Annals of the New York Academy of Sciences, 444, 336-359.

Weingartner, H., Cohen, R.M., Murphy, D.L., Martello, J., & Gerdt, C. (1981). Cognitive processes in depression. Archives of General Psychiatry, 38, 42-47.

Weingartner, H., Grafman, J., Boutelle, W., Kaye, W., & Martin, P.R. (1983). Forms of memory failure. Science, 221, 380-382.

Weingartner, H., Kay, W., Smallberg, S.A., Ebert, M.H., Gillin, J.C., & Sitaram, N. (1981). Memory failures in progressive idopathic dementia. Journal of Abnormal Psychology, 90(3), 187-196.

Weingartner, H., Miller, H., & Murphy, D.L. (1977). Mood-state-dependent retrieval of verbal associations. Journal of Abnormal Psychology, 86(3), 276-284.

Weingartner, H., Sitaram, N., & Gillin, J.C. (1979). The role of the cholinergic nervous system in memory consolidation. Bulletin of the Psychonomic Society, 13(1), 9-11.

Whitehead, A. (1973). Verbal learning and memory in elderly depressives. British Journal of Psychiatry, 123, 203-208.

Whitehead, A. (1974), Factors in the learning deficit of elderly depressives. British Journal of Social and Clinical Psychology, 13, 201-208.

Whitehouse, P.J., Price, D.I., Stuble, R.G., Clark, A.W., Coyle, J.T., & DeLong, M.R. (1982). Alzheimer's disease and senile dementia: Loss of neurons in the basal forebrain. Science, 215, 1237.

Wickelgren, W.A. (1973). The long and the short of memory. Psychological Bulletin, 80(6), 425-438.

Williams, J.M.G., & Scott, J. (1988). Autobiographical memory in depression. Psychological Medicine, 19, 689-695.

Willner, P. (1985). Depression. A psychobiological synthesis. New York: John Wiley & Sons.

Wilson, R.S., Bacon, L.D., Fox, J.H., & Kaszniak, A.W. (1983). Primary memory and secondary memory in dementia of the Alzheimer type. Journal of Clinical Neuropsychology, 5, 337-344.

Wisniewski, K.E., Wisniewski, H.M., & When, G.Y. (1985). Occurence of neuropathological changes and dementia of Alzheimer's disease in Down's syndrome. Annuals of Neurology, 17, 278-282.

Wood, F.B., & Flowers, L. (1988). Cortical activation in psychiatric disorder. In M. Kinsbourne (Ed.), Cerebral hemisphere function in depression (pp. 79-98). Washington: American Psychiatric Press.

Yesavage, J.A., Brink, T.L., Rose, T.L., Lum, O., Huang, V., Adey, M., & Leirer, V.O. (1983). Development and validation of a geriatric depression screening scale: A preliminary report. Journal of Psychiatric Research, 17(1), 37-49.

APPENDIX A

National Adult Reading Test (NART) (1)

CHORD	SUPERFLUOUS
ACHE	SIMILE
DEPOT	BANAL
AISLE	QUADRUPED
BOUQUET	CELLIST
PSALM	FACADE
CAPON	ZEALOT
DENY	DRACHM
NAUSEA	AEON
DEBT	PLACEBO
COURTEOUS	ABSTEMIOUS
RAREFY	DETENTE
EQUIVOCAL	IDYLL
NAIVE	PUERPERAL
CATACOMB	AVER
GAOLED	GAUCHE
THYME	TOPIARY
HEIR	LEVIATHAN
RADIX	BEATIFY
ASSIGNATE	PRELATE
HIATUS	SIDERAL
SUBTLE	DEMESNE
PROCREATE	SYNCOPE
GIST	LABILE
GOUGE	CAMPANILE

(1) Hazel E. Nelson, 1982

APPENDIX B

Mini-Mental State Examination (MMSE) (DIS version) (1)

Let me ask you a few questions to check your concentration and your memory. Most of them will be easy.

1.	What is the year?	Year__________
2.	What season of the year is it?	Season__________
3.	What is the date?	Date__________
4.	What is the day of the week?	Day__________
5.	What is the month?	Month__________
6.	Can you tell me where we are right now? For instance, what city/town are we in?	City/Town__________
7.	What state are we in?	State__________
8.	What are the names of two streets nearby?	Street__________
		Street__________
9.	What floor of the building are we on?	Floor__________
10.	What is this address, or What is the name of this place?	Place__________

11. I am going to name three objects. After I have said them, I want you to repeat them. Remember what they are because I am going to ask you to name them again in a few minutes.

"Apple" "Table" "Penny"

Could you repeat the three items for me? SCORE FIRST TRIAL.

Apple

Table

Penny

INTERVIEWER: REPEAT OBJECTS UNTIL ALL THREE ARE LEARNED.

12. Can you subtract 7 from 100, and then subtract from the answer you get and keep subtracting 7 until I tell you to stop?

COUNT ONLY ONE ERROR IF SUBJECT MAKES SUBTRACTION ERROR, BUT SUBSEQUENT ANSWERS ARE 7 LESS THAN THE ERROR.

(93)

(86)

(79)

(72)

(65)

STOP

13. Now I am going to spell a word forwards and I want you to spell it backwards. The word is "world". W-O-R-L-D. Spell "world" backwards.

REPEAT SPELLING IF NECESSARY.

__ __ __ __ __
W O R L D

14. Now what were the three objects I asked you to remember?

Apple

Table

Penny

15. INTERVIEWER: SHOW WRIST WATCH.

What is this called?

INTERVIEWER: SHOW PENCIL.

What is this called?

16. I'd like you to repeat a phrase after me:

"No if's, and's, or but's"

ALLOW ONLY ONE TRIAL. CODE O REQUIRES AN ACCURATELY ARTICULATED REPETITION.

17. Read the words on this page and then do what it says.

INTERVIEWER: HAND CARD B.

CODE "O" IF RESPONDENT CLOSES EYES.

18. INTERVIEWER: READ FULL STATEMENT BELOW AND THEN HAND RESPONDENT A BLANK PIECE OF PAPER.

DO NOT REPEAT INSTRUCTION OR COACH.

I am going to give you a piece of paper. When I do, take the paper in your right hand, fold the paper in half with both hands, and put the paper down on your lap.

Takes paper in right hand.

Folds paper in half.

Puts paper down on lap.

19. Write any complete sentence on that piece of paper for me.

SENTENCE SHOULD HAVE A SUBJECT AND A VERB AND MAKE SENSE.

SPELLING OR GRAMMATICAL ERRORS ARE OK.

20. Here's a drawing. Please copy the drawing on the same paper.

INTERVIEWER: HAND CARD C.

CORRECT IF TWO CONVEX FIVE-SIDED FIGURES AND INTERSECTION MAKES A FOUR-SIDED FIGURE.

(1) Folstein, Folstein & McHugh, 1975

APPENDIX C

Geriatric Depression Scale (1)

Choose the best answer for how you feel over the best week.

1.	Are you basically satisfied with your life?	Yes/No
2.	Have you dropped many of your activities and interests?	Yes/No
3.	Do you feel that your life is empty?	Yes/No
4.	Do you often get bored?	Yes/No
5.	Are you hopeful about the future?	Yes/No
6.	Are you bothered by thoughts you can't get out of your head?	Yes/No
7.	Are you in good spirits most of the time?	Yes/No
8.	Are you afraid that something bad is going to happen to you?	Yes/No
9.	Do you feel happy most of the time?	Yes/No
10.	Do you often feel helpless?	Yes/No
11.	Do you often get restless and fidgety?	Yes/No
12.	Do you prefer to stay at home, rather than going out doing new things?	Yes/No
13.	Do you frequently worry about the future?	Yes/No
14.	Do you feel you have more problems with memory than most?	Yes/No
15.	Do you think it is wonderful to be alive now?	Yes/No
16.	Do you often feel downhearted and blue?	Yes/No
17.	Do you feel pretty worthless the way you are now?	Yes/No
18.	Do you worry a lot about the past?	Yes/No
19.	Do you find life very exciting?	Yes/No
20.	Is it hard for you to get started on new projects?	Yes/No

21.	Do you feel full of energy?	Yes/No
22.	Do you feel that your situation is hopeless?	Yes/No
23.	Do you think that most people are better off than you are?	Yes/No
24.	Do you frequently get upset over little things?	Yes/No
25.	Do you frequently feel like crying?	Yes/No
26.	Do you have trouble concentrating?	Yes/No
27.	Do you enjoy getting up in the morning?	Yes/No
28.	Do you prefer to avoid social gatherings?	Yes/No
29.	Is it easy for you to make decisions?	Yes/No
30.	Is your mind as clear as it used to be?	Yes/No

(1) Yesavage et al. (1983)

APPENDIX D

Experimental Stimuli from the Categorised and Noncategorised Word Lists

	Categorised (related words)		Noncategorised Words (unrelated words)	
	Word Frequency (1)	Degree of Association to Category Name (2)		Word Frequency (1)
Category: FRUITS				
apple	9	0.97	cheese	9
orange	23	0.88	farmer	23
banana	4	0.64	chisel	4
pear	6	0.74	robe	6
Category: CLOTHING				
shirt	27	0.80	cloud	28
socks	7	0.75	glen	7
pants	9	0.72	comic	9
coat	43	0.59	weapon	42
Category: A PART OF THE HUMAN BODY				
legs	67	0.90	chair	66
arms	121	0.90	radio	120
head	424	0.70	water	442
eye	122	0.69	game	123
Category: ANIMALS				
dog	75	0.96	judge	77
cat	23	0.93	craft	23
horse	117	0.79	price	108
cow	29	0.64	tea	28

(1) Kucera and Francis (1967)

(2) Battig and Montague (1969)

APPENDIX E

<u>Experimental Stimuli of Presentation of the Five Learning Trials as given to Subjects</u>

RELATED/CLUSTERED WORD LIST — A

TRIAL 1:	TRIAL 2:	TRIAL 3:	TRIAL 4:	TRIAL 5:
APPLE	SHIRT	LEGS	DOG	APPLE
ORANGE	SOCKS	ARMS	CAT	ORANGE
BANANA	PANTS	HEAD	HORSE	BANANA
PEAR	COAT	EYE	COW	PEAR
SHIRT	LEGS	DOG	APPLE	SHIRT
SOCKS	ARMS	CAT	ORANGE	SOCKS
PANTS	HEAD	HORSE	BANANA	PANTS
COAT	EYE	COW	PEAR	COAT
LEGS	DOG	APPLE	SHIRT	LEGS
ARMS	CAT	ORANGE	SOCKS	ARMS
HEAD	HORSE	BANANA	PANTS	HEAD
EYE	COW	PEAR	COAT	EYE
DOG	APPLE	SHIRT	LEGS	DOG
CAT	ORANGE	SOCKS	ARMS	CAT
HORSE	BANANA	PANTS	HEAD	HORSE
COW	PEAR	COAT	EYE	COW

APPENDIX F

<u>Experimental Stimuli from the Interference Lists (List B) for Related and Unrelated Word Lists</u>

RELATED WORD LIST	UNRELATED WORD LIST
DESK	WEATHER
RANGER	PARENT
HOUSE	ROSE
MOON	SOAP
STOVE	SCHOOL
MOUNTAIN	NEST
GLASSES	CALENDAR
RIVER	TYPEWRITER
BELL	LOCK
BOAT	CANDLE
COFFEE	FIGHT
GUN	KNIFE
PENCIL	BOX
CHURCH	JAR
GARDEN	MEDAL
COLOUR	TUNNEL

APPENDIX G

Cued Recall of Related Words

QUESTION: From the first list of words I read out to you, can you recall any words that were ....?

FRUITS:	APPLE, ORANGE, BANANA, PEAR
CLOTHING:	SHIRT, SOCKS, PANTS, COAT
PARTS OF THE BODY:	LEGS, ARMS, HEAD, EYE
ANIMALS:	DOG, CAT, HORSE, COW

APPENDIX H

Words Presented in Recognition Trial for the Related Word List

MASK	APPLE
EYE	BED
COT	SALARY
SHIRT	GRAIN
DECK	CAT
HEAD	SET
LEGS	BLOCK
HOOK	BANANA
RULE	DOG
WOUND	COW
PANTS	GREASE
PEAR	DISC
CHART	ORANGE
DEGREE	ARMS
HORSE	LENGTH
SOCKS	ABBEY

APPENDIX I

Recognition Trial Word Frequency — Related-Categorised Word List

Categorised (related words	Word Frequency (1)	Recognition Words	Word Frequency (1)
APPLE	9	MASK	9
ORANGE	23	CHART	22
BANANA	4	HOOK	5
PEAR	6	DISC	6
SHIRT	27	GRAIN	27
SOCKS	7	ABBEY	7
PANTS	9	GREASE	9
COAT	43	SALARY	43
LEGS	67	BLOCK	66
ARMS	121	DEGREE	128
HEAD	424	SET	414
EYE	122	BED	127
DOG	75	RULE	73
CAT	23	DECK	23
HORSE	117	LENGTH	116
COW	29	WOUND	28

(1) Kucera and Francis (1967)

APPENDIX J

Experimental Stimuli of Presentation of Learning Trials of Unrelated Words given to Subjects

UNRELATED WORD LIST — LIST A

TRIAL 1:	TRIAL 2:	TRIAL 3:	TRIAL 4:	TRIAL 5:
CHEESE	CLOUD	CHAIR	JUDGE	CHEESE
FARMER	GLEN	RADIO	CRAFT	FARMER
CHISEL	COMIC	WATER	PRICE	CHISEL
ROBE	WEAPON	GAME	TEA	ROBE
CLOUD	CHAIR	JUDGE	CHEESE	CLOUD
GLEN	RADIO	CRAFT	FARMER	GLEN
COMIC	WATER	PRICE	CHISEL	COMIC
WEAPON	GAME	TEA	ROBE	WEAPON
CHAIR	JUDGE	CHEESE	CLOUD	CHAIR
RADIO	CRAFT	FARMER	GLEN	RADIO
WATER	PRICE	CHISEL	COMIC	WATER
GAME	TEA	ROBE	WEAPON	GAME
JUDGE	CHEESE	CLOUD	CHAIR	JUDGE
CRAFT	FARMER	GLEN	RADIO	CRAFT
PRICE	CHISEL	COMIC	WATER	PRICE
TEA	ROBE	WEAPON	GAME	TEA

APPENDIX K

Words Presented in Recognition Trial — Unrelated Word List

HYMN	CHEESE
GAME	LEAD
WEAPON	TOUR
CLOUD	MIRROR
GIANT	CRAFT
WATER	NIGHT
CHAIR	METAL
PIRATE	CHISEL
VOTE	JUDGE
FROZEN	TEA
COMIC	LATENT
ROBE	LASH
HABIT	FARMER
SPRING	RADIO
PRICE	IMAGE
GLEN	SHRINE

APPENDIX L

Recognition Trial Word Frequency — Unrelated Word List

Non categorised (unrelated words)	Word Frequency (1)	Recognition Words	Word Frequency (1)
CHEESE	9	HYMN	9
FARMER	23	HABIT	23
CHISEL	4	PIRATE	4
ROBE	6	LASH	6
CLOUD	28	MIRROR	27
GLEN	7	SHRINE	7
COMIC	9	LATENT	9
WEAPON	42	TOUR	43
CHAIR	66	METAL	61
RADIO	120	SPRING	127
WATER	442	NIGHT	411
GAME	123	LEAD	129
JUDGE	77	VOTE	75
CRAFT	23	GIANT	23
PRICE	108	IMAGE	119
TEA	28	FROZEN	27

(1) Kucera and Francis (1967)

APPENDIX M

Simple Main Effects of Univariate Analysis of Variance Summary Table for Demented, Depressed and Control Groupswith Related versus Unrelated Words and Learning Trials

	SS	df	MS	F
Demented Group	768.80	1	768.80	100.03*
words	33.80	1	33.80	8.70***
trials	23.83	4	5.96	2.67
words by trials	5.83	4	1.46	0.75
Depressed Group	2976.80	1	2976.80	60.02*
words	151.25	1	151.25	58.98*
trials	136.83	4	34.21	13.09*
words by trials	20.37	4	5.09	2.44
Control Group	4512.50	1	4512.50	16.45*
words	273.78	1	273.78	33.27**
trials	151.40	4	37.85	13.22*
words by trials	2.52	4	0.63	0.18

* p<.000   ** p<.001   *** p<.01

APPENDIX N

Simple Main Effects of Univariate Analysis of Variance Summary Table for Demented, Depressed and Control Groups with Related Words Recalled in the Form of Semantic Clusters

	SS	df	MS	F
Demented Group	184.90	1	184.90	90.51
Within Subjects	49.20	28	1.76	
Trials	1.60	4	0.40	0.23
Depressed Group	819.03	1	819.03	56.67
Within Subjects	66.45	28	2.30	
Trials	66.35	4	16.59	7.21*
Control Group	1713.96	1	1713.96	80.62*
Within Subjects	47.36	16	2.96	
Trials	52.64	4	13.16	4.45***

* p<.000   *** p<.01

APPENDIX O

Simple Main Effects of Univariate Analysis of Variance Summary Table for Demented, Depressed and Control Groups with Related Words with Retrieval Processes

	SS	df	MS	F
Demented Group	1365.04	1	1365.04	256.23*
Within Subjects	37.29	7	5.33	
Retrieval Processes	524.33	2	262.17	65.15*
Depressed Group	3082.67	1	3082.67	157.13*
Within Subjects	137.33	7	19.62	
Retrieval Processes	254.08	2	127.04	25.44*
Control Group	3024.60	1	3024.60	285.34*
Within Subjects	42.40	4	10.60	
Retrieval Processes	29.20	2	14.60	3.17

* p<.000